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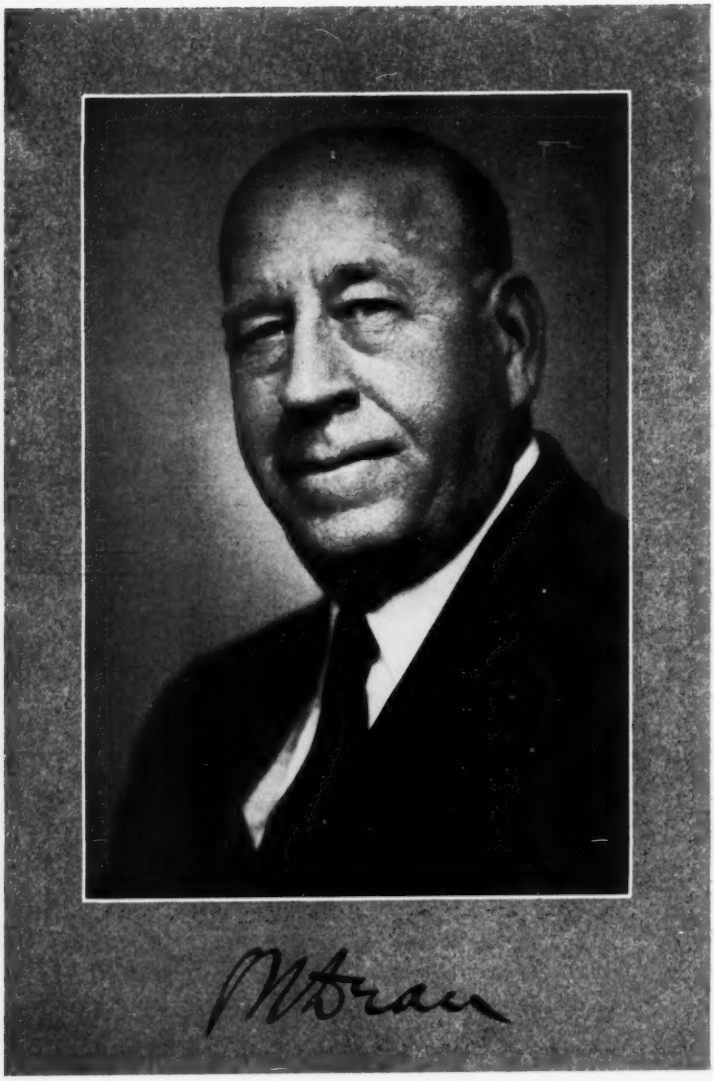

LEE WALLACE DEAN, SR., M.D.

1873-1944

Dr. Lee Wallace Dean, Sr., Co-Editor of the ANNALS, died at his home near St. Louis on February 9, 1944.

Born in Muscatine, Iowa, March 28, 1873, he received his early education in that state and subsequently attended the State University where the degree of Bachelor of Science was conferred upon him in 1894. The degrees of Master of Science and Doctor of Medicine followed in 1896.

After a year of graduate work in Vienna, Dr. Dean returned to Iowa and three years later was made Head of the Department and Professor of Otolaryngology and Oral Surgery. In 1912 he became Dean of the College of Medicine, and held both of these positions until 1927 when he came to Washington University as Head of the Department and Professor of Otolaryngology in the School of Medicine and Laryngologist to the hospitals associated with this institution. Following his retirement in 1940 he was made Emeritus Professor.

During the First World War he served as commanding officer of General Hospital No. 54, with the rank of Lieutenant-Colonel.

Dr. Dean was President of the American Laryngological Association in 1924; of the American Otological Society in 1932; of the American Laryngological, Rhinological and Otological Society in 1937. He was a Fellow also of the American Broncho-Esophagological Association, the American College of Surgeons, the American Medical Association and the Societé de Laryngologie des Hôpitaux de Paris.

In 1937 the American Laryngological Association awarded him its de Roaldes medal, the most distinguished honor which a laryngologist may attain.

At the time of his death he was still an active member of the American Board of Otolaryngology.

Dr. Dean will be remembered chiefly as an educator, for his every thought revolved about the development of young men in the specialty and the improving of standards in the teaching of otolaryngology.

He and Mrs. Dean were ardent hunters and fishermen and the season was invariably crowned by a series of sumptuous dinners in honor of their many friends.

Dr. Dean is survived by his widow, the former Ella Bailey, and a son, Dr. Lee Wallace Dean.

* * *

In the passing of Dr. Dean the ANNALS loses a lifelong friend and valued adviser. As a mark of profound respect to its departed Editor an early issue will be dedicated to his memory, which will include an appreciation of his life and works, together with a collection of scientific articles by his former colleagues.

ANNALS
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I

OTITIS EXTERNA

WILLIAM H. JOHNSTON, M.D.

SANTA BARBARA, CAL.

Inflammatory conditions of the external auditory canal and the pinna are responsible for about one-third of the cases seen by the otologist. This is especially true of the southern coastal areas on account of year-round ocean bathing. In spite of their complicated appearance, the external ear and the auditory canal have a very simple histologic structure, which consists of skin, cartilage, and bone. Being chiefly skin, the external ear is subject to all skin diseases and skin affections. We may find psoriasis, pyoderma, lupus erythematosus, granuloma annulare, drug eruptions and poison ivy, among many other conditions.

Indeed, the external ear belongs as much in the domain of the dermatologist as in that of the otologist, the only difference being in the point of view and the nomenclature. The otologist refers to dermatitis as an otitis externa. We are indebted to the dermatologist for much of our knowledge of the etiology and treatment of these affections.

Generally, all cases and all types of otitis externa are the result of four or five factors such as external irritants, systemic diseases

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Revision of a paper read before the Eye, Ear, Nose and Throat Section of the California Medical Association held at Del Monte, Calif., May 5-8, 1941.

which produce a special predisposition to skin affections, general and local allergy or some interference with the normal secretion of cerumen. A lack of cerumen is found in a vast majority of cases of external otitis. Alfödy[1] stressed the importance of focal infection in many types of external otitis. The pathogenic organisms found are staphylococci, pyocyaneus, streptococci, Friëdlander's, diphtheria and tubercle bacilli. It is believed that the cerumen itself has bactericidal properties and that streptococci and diphtheria bacilli are killed by it in a few days. The inflammation may also be caused by a number of fungi and by the irritation of various parasites and artificially induced substances.

There are various classifications of external otitis and the one used in this discussion is, for the most part, based on the pathology.

1. Circumscribed inflammation and furuncle.
2. Diffuse inflammation.
3. Impetiginous dermatitis.
4. Eczema or seborrheic otitis externa.
5. Artificial otitis externa.
6. Hemorrhagic otitis externa.
7. Otomycosis.
8. Perichondritis.
9. Chondrodermatitis nodularis chronica helicis.
10. Periostitis.

CIRCUMSCRIBED INFLAMMATION

The most frequent external ear affection is the circumscribed type of inflammation such as is seen in the ordinary furuncle of the external canal. A furuncle may occur at any part of the ear canal containing hair follicles. In adults, only the outer third of the meatus normally contains hair follicles but in children there may be hair as far inward as the tympanic membrane, and they are, therefore, subject to deep furuncles. English military surgeons say that it is the most common inflammation of the canal seen in military service. Furuncles may be entirely primary but often they are preceded by boils on other parts of the body. Scaly dermatitis or chronic focal infection predisposes to this condition. It most frequently occurs in hot weather; more often in those who sea bathe and in those who are particularly addicted to cleaning out their ears after bathing with the corner of a towel, a handkerchief or a match stick.

The suggestive symptoms of a developing furuncle are a feeling of tightness followed by pain which may be spontaneous or felt

only on mastication or swallowing. There is usually an intermittent deafness and sometimes a little discharge. The temperature and the pulse rate may be as high as in mastoiditis. It is sometimes quite difficult to discover an early furuncle in the meatus but pain on any motion of the auricle is a helpful sign.[3] A tender and palpable gland or the presence of lymphangitis with edema over the mastoid may result from a furuncle in the posterior meatal wall. There is usually swelling of the meatus and the surrounding tissues obstructing a view of the tympanic membrane; often pus can be seen coming from a pin-point opening in the cartilaginous portion. Hearing tests show that air conduction is reduced and bone conduction is relatively increased.[11] Although one should be able to differentiate furunculosis from mastoiditis,[2] it should not be forgotten that the two diseases may be associated and their presence may be very difficult to recognize. We can all recall instances that would illustrate the above point.

The treatment of ear furuncle is not yet standardized. There is much controversy as to the value of early incision. Politzer recommended incision, Guttich[7] and others condemned it entirely, while a third group recognized the necessity of incision in some cases for the relief of pain.[17] This procedure may be carried out with or without general anesthesia. Local anesthetic seems to have little effect in relieving the pain caused by the incision. The incision is made from within outward but one should not always expect to find pus. It is recommended that after incising, the canal be loosely packed with gauze soaked in a germicidal solution.[11] Some observers[20] recommend scarifying the skin on the inflamed area allowing the blood to fill the canal and remain, thereby using a type of autohemotherapy.[7] Early furuncle may be aborted by the use of x-ray; others use strips of gauze or tampons of cotton saturated with glycerin containing five per cent phenol. Solution of aluminum acetate N. F. has been the most satisfactory in my hands.

The pain of furunculosis is often very severe and its relief is a real problem. The soft tissue of the external canal is quite compact and the collection of a small amount of inflammatory exudate in the tissues results in very severe pain. A tight-fitting plug of cotton, saturated with metacresylacetate, preventing the stretching of the tissue has, in my hands, given the greatest amount of relief. For the relief of pain, good results are obtained from the local use of heat; this may be with hot moist compresses, electric pad, infra red rays or radiothermy; also aspirin, phenacetin or codeine taken internally are helpful. For general supportive treatment, cod liver oil, autogenous vaccine and chemotherapy may be employed. One observ-

er[10] recommended the use of autohemotherapy, administering 10 cc. of whole blood subcutaneously every third day. The earlier the treatment is instituted, the better the results.

In recurring furuncle, one should search for foci of infection. Boils have a tendency to recur even after a lapse of years without any apparent exciting cause. Therefore, it is advisable to sterilize and harden the lining of the meatus with drops of salicylic acid in alcohol following an acute inflammation.

DIFFUSE INFLAMMATION

Diffuse inflammation of the meatus occurs mostly following the circumscribed variety. The bacteria rarely come from the outside but frequently from the discharge of some middle ear disease. The infection gains entry through fissures of the skin or through trauma from scratching. It is usually a staphylococcal or streptococcal infection,[22] and sometimes pyocyaneus, Friëdlander's, diphtheria bacilli or Vincent's organisms may be found. This is the type of external otitis which often ends in erysipelas and spreads to the auricle and the surrounding scalp. There is fever, acute pain, some itching and a watery discharge. The meatus is swollen and the tympanic membrane may be reddened. This type may become very serious and everything should be done to prevent it. The best method of prevention is proper ear hygiene consisting chiefly in the avoidance of too much meddling with the ear.

If the inflammation is secondary, the cause should be first removed. Treatment of the infection should be the local use of antiseptics either in oily solutions or in the form of insufflations.

Preparations recommended are: Phenyl mercuric nitrate 1 to 25 in alcohol with 1% castor oil,[6] silver nitrate 2% to 5%, 1 to 2000 flavine in glycerin, and solutions of lead and aluminum acetate. One observer reports excellent results from the use of 1% brilliant green in all types of external otitis. Another recommends Bonney's paint which is a mixture of brilliant green and crystal violet. If the auricle is also involved, tar and ichthyol lotions may be useful. In streptococcal infections, sulfanilamide should be given orally. Various vaccines and foreign protein injections have been used successfully by some observers. For a spreading erysipelas, chemotherapy gives the best results.

IMPETIGINOUS DERMATITIS OF THE EXTERNAL CANAL

This type is seen mostly in children. It is a streptococcal infection almost always secondary to otitis media or to impetigo elsewhere on the body. Its treatment should be directed: 1) Against the impetigo, using ointment of ammoniated mecury 2% to 5%. 2) Against the otitis media and should include supportive measures such as ultraviolet irradiation and the use of sulfonamides.

ECZEMA OR SEBORRHEIC OTITIS EXTERNA

This may occur in acute or chronic form and constitutes about 30 per cent of all diseases of the external ear.[19] It has been stated that it is practically always secondary to allergy and is aggravated sometimes by the irritating discharge coming from an infected middle ear.[24] Anything that interferes with the proper ventilation of the meatus predisposes to this type of external otitis. The wearing of headphones, the use of binaural stethoscopes are usual factors. Frequent syringing and the consequent excessive formation of wax, frequent diving, and the use of hydrogen peroxide are considered other predisposing factors. There is some pain around the ear, intolerable itching, and if the meatus becomes blocked,—deafness. The meatus may be swollen, red and covered with scaly formations. (Discharge may be profuse). The condition may spread to the auricle, causing swelling, the formation of vesicles and later extensive fissures. All the diseased skin is open for entry of either staphylococci or streptococci causing a diffuse inflammation and even erysipelas. The desquamative type of eczema is at times responsible for the development of primary cholesteatoma of the auditory meatus. In many cases of eczema of the ear we find an infectious focus in Rosenmüller's fossa or in one of the paranasal sinuses.

The treatment of eczema is rather difficult. If there is a primary condition which is responsible, it should be corrected first. Mastoidectomy, tonsillectomy, eradication of a nasal sinus infection may be necessary. The local treatment is symptomatic; antiseptic substances in solution or in powder have been used against the secondary infection. Radiotherapy has been recommended for the relief of itching.[9] Others advise the use of iodine, phenol and alcohol, thorium X ointment,[9] ionized zinc, mild oils, ointment of salicylic acid and sulphur. A mild hypothyroidism may be found associated with this type and, in several cases, the writer has found a local improvement from the use of thyroid extract. Water in general should not be allowed to come in contact with the affected parts. The diet should be carefully investigated and regulated as indicated.

ARTIFICIAL OTITIS EXTERNA

This condition is seen frequently in the military service. In the past, it has been found that soldiers have used all kinds of substances to produce what appeared to be a serious otitis media. In the Moroccan army they mixed vinegar and table salt[16] which, when put into the meatus, caused a very profuse otorrhea. In the first World War, cases were reported where a mixture containing limburger cheese was placed in the ear canal to simulate a serious chronic ear disease. The diagnosis is not difficult because such a serious looking affection was not preceded by pain, fever or infection in the nose or throat. Foreign bodies introduced into the canal by small children are a common source of this type of external otitis. Paper clips, small glass beads, pits from cherries, pencil erasers, kernels of corn and many other items have been found.

HEMORRHAGIC OTITIS EXTERNA

This type is usually secondary to an otitis media and is seen quite often during influenza epidemics. The pain is severe and stabbing in character and there is tinnitus and deafness. One or more blebs may be seen on the membrane covering the osseous portion of the meatus, usually on the roof or the posterior wall. The bleb may be so large as almost to obscure the view of the tympanic membrane. In such cases, the primary otitis media should be treated. If the ear drum is not perforated, the blebs should be opened. This may be done with a myringotomy knife or, many times, all that is necessary is to rupture them by pressure with a cotton-tipped applicator.

OTOMYCOSIS

This is one of the most frequent inflammatory affections of the external auditory canal and was formerly thought to be a rare disease. The usual fungi causing it are the aspergillus niger, mucor, eurotium, sterigmatocystis; the latter is frequently mistaken for aspergillus. Rarely one meets with severe external otitis caused by actinomyces or streptothrix. In the milder mycotic diseases there is some pain, itching, irritation and a thin discharge having a musty odor. There is slight deafness due to blocking of the canal and the ear drum is red and edematous. Painful swelling in the pretragal lymph nodes is often noted. On inspection of the ear, one sees a mass resembling wet blotting paper which obscures the view of the tympanic membrane. When the debris is removed, it reforms quite rapidly. The diagnosis is made microscopically. In cases of aspergillus niger the black pigment will aid in the making of a correct diagnosis.

For the treatment of this condition a great many substances have been recommended: alcohol solutions of salicylic acid, tincture of iodine[21] copper sulphate, absolute alcohol, 10% solution of crystal violet, metacresylacetate and thymol.[18] In my hands, use of a cotton tampon soaked in metacresylacetate, left in the canal for 24 hours and followed by a daily application of 2% thymol in metacresylacetate has been the most satisfactory. Some authors recommend the use of 2% thymol in alcohol and metacresylacetate. Thymol in metacresylacetate is much less painful than the latter preparation. Whalen[23] finds the use of thymol iodide dusted into the canal three times a day for three days after the above treatment is helpful. He also advises the administration of 30 grains of potassium iodide daily for 30 days.

Cases of fungus infections of the canal should be watched for a couple of months as some remaining spores may set up an exacerbation. In fungus infections, the sulfonamides do not have any appreciable effect regardless of whether they are used locally or internally. As a matter of fact, I have seen one quite severe local reaction from the use of sulfathiazole powder. Occasionally one may see cases of external ear inflammation caused by the presence of larvae and maggots in the meatus;[15] the most frequent cause of myiasis is the lucilia sericata. The maggots cause the earache and the discharge. The larvae may completely obstruct the meatus setting up an inflammation of the canal wall and the tympanic membrane. The treatment is not difficult.

PERICHONDRITIS OF THE EXTERNAL CANAL AND EAR

When the cartilage of the external ear is also inflamed, we speak of a chrondritis or perichondritis.[20] It may be caused by simple bacteria, mostly pyocyaneus, and may be preceded by chemical or mechanical injury of the external ear.[13, 18] The infection may occur in hematoma or after cutting a meatal flap in radical mastoid operations. Perichondritis results in much swelling and often in abscess formation with subsequent shrinking and deformity.

In the early stages, the treatment consists of fomentations and removal of the pack from the mastoid cavity. If there is a definite softening in any part of the swelling, immediate incision is indicated, completely through the auricle. A rubber drain is inserted in order to prevent localized infection and consequent necrosis of cartilage. For a staphylococci perichondritis, a small incision is suggested but for a pyocyaneus infection a large incision is necessary; chemotherapy is

always indicated. X-ray treatment is indicated in all stages; if applied early, it may abort the inflammation. A dose of 30 R will give almost complete relief of pain.

CHONDRODERMATITIS NODULARIS CHRONICA HELICIS

A specific type of chondritis is the one named chondrodermatitis nodularis chronica helicis.[4, 8, 12, 14] It was first diagnosed by Winkler in 1915. It is a harmless, common ailment chiefly seen in telephonists, soldiers, nuns and physicians. It is also known as keratoma auriculare and angiokeratoma. The nodule is hard, small, immovable and surrounded by a hyperemic area. The site of the disease is usually the upper pole of the auricle and is sometimes bilateral. It is seen most often in elderly persons. The condition is characterized by tenderness and interrupted attacks of pain. There are inflammatory changes in the nodules which consist of granulation tissue, acanthosis of the epidermis, degeneration of the cartilage and signs of perichondritis. This condition is caused probably by trauma, either chemical or mechanical. There is always danger of secondary infection. The treatment which is at present accepted is incision and removal of the diseased cartilage with curette. These nodules have a tendency to recur.

PERIOSTITIS

Periostitis of the meatus is the cause of exostosis sometimes seen in divers, the exact cause being unknown.[5] Many predisposing factors may precipitate such a periostitis. The exostotic mass sometimes obliterates the meatus and causes deafness. According to recent observers,[12] the exostosis predisposes to a sudden rupture of the tympanic membrane while diving. Such a rupture is extremely dangerous and may result in drowning. The exostosis may be dealt with by surgical methods, the best being the removal with burrs propelled by an electric dental drill.

SUMMARY

In the treatment of external otitis, the remedies recommended are so numerous that no real attempt can be made to enumerate them. Many of them are without much, if any, effect. Metacresylacetate is both germicidal and anesthetic and is our most useful remedy. For the simple cases of otitis externa, an ointment composed of phenol, percipitated sulphur and salicylic acid 9 grains each to 1 ounce of an animal oil base called Neatsol is suggested. No definite routine can be followed in the treatment of otitis externa. However, the pro-

cedure of treatment in any type of external ear inflammation is:

1. Thorough cleansing.
2. Removal of the cause if it can be ascertained.
3. Use of germicides.
4. Protection of the inflamed part.
5. General supportive measures.

The large number of cases of otitis externa seen in persons who have been bathing in public plunges suggests the necessity of a more careful check on these pools by our public health departments.

1515 STATE STREET.

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II

RELATIONSHIP OF POLIOMYELITIS AND TONSILLECTOMY

ROBERT E. HOWARD, M.D.

CINCINNATI, OHIO

In accepting the invitation to give the following report of the relationship between tonsillectomy and poliomyelitis, I admit that I accepted in the hope of clarifying my own views on the subject so that I might truthfully answer the often asked question, "Will my child get poliomyelitis if you take out his tonsils and adenoids during the poliomyelitis season?"

This question is no doubt asked pediatricians even more than throat specialists, and with only few exceptions, I believe both of these groups are as confused as I have been in knowing what is the correct answer.

Widespread publicity of the excellent paper and the complete survey of the K. family by Toomey, Krill and others probably has been the greatest factor in crystallizing public knowledge of the occasional serious relationship between tonsillectomy and poliomyelitis. These authors as well as many others have shown from their reports in the literature that there is a real hazard when poliomyelitis does complicate a simple tonsil and adenoid operation.

As early as 1910 and 1912 Sheppard and Boyd pointed to this operation during the poliomyelitis season with caution. It was sixteen years later that Ayer directed our attention to the occurrence of bulbar poliomyelitis in nine cases following tonsillectomy and adenoidectomy. In 1929 Aycock and Luther reported the largest series up to that time, 16 cases of poliomyelitis following tonsil and adenoid operations. This series consisted of 7 of the bulbar type, 5 of the bulbo-spinal, and 4 of the spinal. It is interesting to note that this group was taken from 714 patients with poliomyelitis in Boston, Mass., and of this group 217 or 30% had had their tonsils and adenoids removed—36 within the year and 16 of the 36 within 7 to 18 days preceding the onset of poliomyelitis.

Presented before the Midwestern Section Meeting of the American Laryngological, Rhinological and Otological Society, Cleveland, Ohio, Jan. 12, 1944.

In very recent correspondence, Dr. W. L. Aycock tells me he is very gratified from the standpoint of preventive medicine that the views expressed in his 1929 paper have now been confirmed by subsequent reports. He also stated in his letter, "The practice of postponing tonsillectomy in the presence of an outbreak of poliomyelitis is now becoming common." This was substantiated by a letter from Dr. Charles F. Ferguson in charge of the Ear, Nose and Throat Service at the Boston Children's Hospital where they banned all tonsillectomies and adenoidectomies from August 15 to November 5, 1943, because of epidemics in all parts of the country. During this period five cases of bulbar poliomyelitis were reported and observed following recent tonsil and adenoid removal, but none were resultant from operations in that hospital.

It was at the Boston Children's Hospital that Eley & Flake made their revealing study of the predominance of the bulbar type in 29 cases following tonsillectomy and adenoidectomy in 1938. Fischer, Stillerman and Marks have also been interested in this study and have shown that the spinal type predominates if poliomyelitis develops within 30 to 60 days following tonsillectomy and adenoidectomy. The banning of these operations in most of the hospitals of Chicago and Cook County during August and September of 1943 due to the poliomyelitis epidemic in that area was initiated by Drs. Bundeson and Piszczek, and probably prevented many cases from developing.

I have been unable to determine from statistics already in the literature what is the percentage of chance of developing poliomyelitis following nose and throat operations during the poliomyelitis season in nonepidemic years.

In Cincinnati we have probably been quite fortunate not to have had a serious epidemic for many years. Thus I will attempt to compute the incidence of poliomyelitis following recent tonsillectomy in nonepidemic years to the number of tonsillectomies and adenoidectomies done during those years in the hospitals only, as this will give the most conservative estimate. (Office tonsillectomies and adenoidectomies excluded.)

During the seven-year period from 1937 to 1943 inclusive, there were 36,295 tonsil operations performed in the hospitals of Cincinnati, of which 23,442 were tonsil and adenoid operations on children of the age most susceptible to poliomyelitis. This averages 5,420 tonsillectomies, or 3,348 tonsillectomies performed in the hospitals per year of a population of approximately 500,000. Of this 3,348

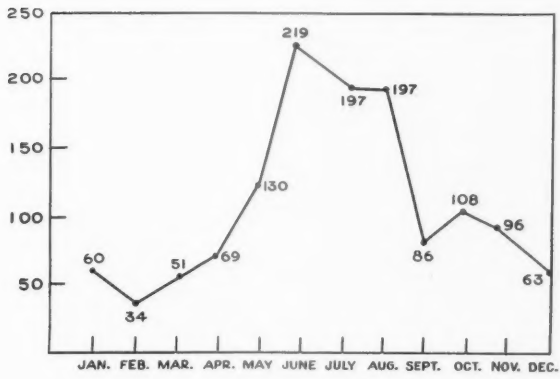

Fig. 1.—Average monthly number of tonsil and adenoid operations Children's Hospital, 1937-1943, Cincinnati, Ohio.

an average of 1,851 tonsillectomies and adenoidectomies are done yearly in Cincinnati during the months when poliomyelitis occurs—July, August, September, and October.

At the Children's Hospital in Cincinnati where only tonsillectomies and adenoidectomies are done, Fig. 1 shows the average monthly operations during the years 1937 to 1943.

July, August, September and October are the popular months for tonsillectomies and adenoidectomies because of vacations and freedom of the patient from upper respiratory infection; in the remaining eight months one postpones at least one-third of the operations on account of colds, sore throats, and ear infections. The question therefore arises, which is the more dangerous in years in which there is no epidemic of poliomyelitis, to take the risk of a tonsillectomy and adenoidectomy during months when poliomyelitis most frequently occurs, or to postpone the operation to months when poliomyelitis is minimal and colds are prevalent?

During the seven years tabulated in Cincinnati hospitals, there has been 257 cases of poliomyelitis of which 233 have occurred in the four months of July, August, September and October, and the remaining 24 cases mostly in November. Of this group, only 6 cases of poliomyelitis developed following recent tonsillectomies and adenoidectomies in Cincinnati.

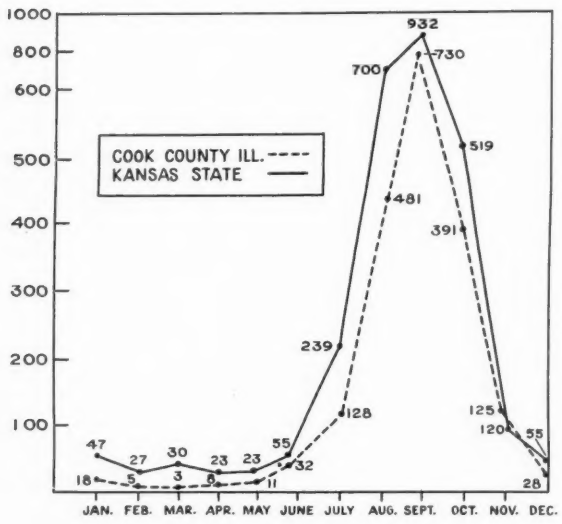

Fig. 2.—Cook County, Ill., total poliomyelitis cases by months, 1932-1943; Kansas State total poliomyelitis cases by months, 1921-1943.

The tonsillectomies and adenoidectomies were performed 10 to 21 days previous to the development of initial symptoms of poliomyelitis. Three spinal and three bulbar cases resulted, with one bulbar death, and five recoveries with minimal paralysis. Five of the six operations were done early in July of 1937, 1939 and 1942, before one realized the presence of the virus in the locality and thus probably these cases could not have been avoided. However, counting the entire group the approximate ratio of poliomyelitis to tonsillectomies and adenoidectomies performed in July, August, September and October is 1 to 2000, and to the total number of cases of poliomyelitis during those months 1 to 40. This is practically the same ratio as Aycock and Luther give in the 16 cases of poliomyelitis following tonsillectomy and adenoidectomy in a total of 714 poliomyelitis cases, or a ratio of 1 to 44.

In selecting the months July, August, September and October as the months in which poliomyelitis most frequently occurs I believe Figs. 2 and 3 will show that most sections of the country have their greatest number of cases during these months. The only exceptions are the states of California and Texas, where poliomyelitis starts generally in June and lasts through November in most years studied.

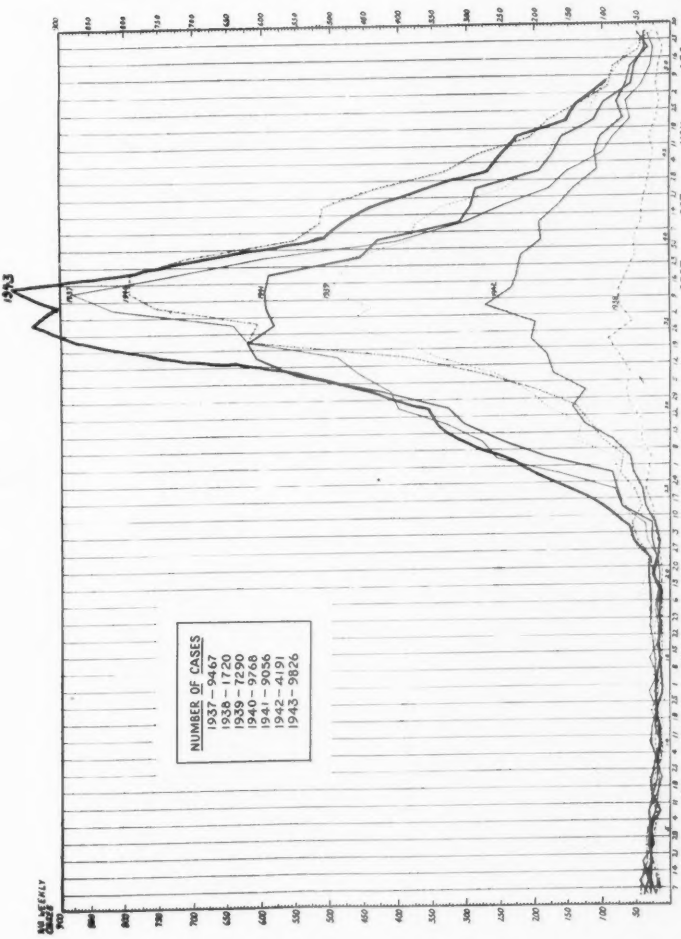

Fig. 3.—Poliomyelitis in the United States, 1937-1943.

Thus the relationship of poliomyelitis to tonsillectomy and adenoidectomy should be based on the number of such operations performed during these months when the virus may be present either in the throat at the time of operation or afterwards.

As previously shown in Table 1, many authors since 1910 have demonstrated the relationship between recent tonsillectomy and adenoidectomy and the bulbar type poliomyelitis. This was simulated in the laboratory by Sabin by observations on the monkey in 1938. He injected the virus in the tonsillo-pharyngeal region and found this region was especially susceptible, since 80% of the monkeys developed the disease while none of those inoculated with the same dose and virus subcutaneously developed poliomyelitis.

A clinical picture of this was presented to me in a communication from Dr. H. L. Wynns of the California State Department of Public Health. "A mother and child from out of the state moved to a home of friends on July 1, 1943. On July 5, the child became ill and died of bulbar poliomyelitis on July 10. The two children in the home visited had their tonsils and adenoids removed on July 6, the day after the visiting child became ill and these children expired on July 17 and 20 respectively of bulbar poliomyelitis."

The reverse of this case was cited in a communication received recently from Dr. A. Piszczek, Health Officer of Cook County Public Health Department. "A white male, aged 3, had a tonsillectomy and adenoidectomy on July 20, 1943, and came down with bulbar poliomyelitis on August 2, 1943, thirteen days later. He had difficulty in swallowing and talking on August 4 and expired on August 8, 1943. A 6-year-old sister of this patient came down with bulbar poliomyelitis on August 14, 1943." These two groups of cases certainly demonstrate direct contact with the virus and the possibility, as shown in the last case, of specificity of virulent strains of the virus, whether a tonsillectomy and adenoidectomy has been performed or not.

As early as 1911 Flexner and Clark recognized different strains of the virus and in 1931 Burnet and MacNamara demonstrated immunological differences between different strains. In 1913 Wickman recognized abortive cases, while Kling and Flexner discovered many healthy carriers through monkey inoculations with the virus obtained from throats of normal individuals.

The organism causing poliomyelitis is a filtrable virus and may be present on any surface of the gastro-intestinal tract from the lips to the lower bowel of persons afflicted with the disease, convalescent,

or associating with carriers. The incidence of cases in widely separated parts of the nation with no particular connection indicates that the virus is widely distributed among normal people and manifests itself only in those who are susceptible. The infection may be transferred by the hands, handkerchiefs, towels, and other recently handled articles of so-called "carriers" or of persons having the disease. It may also spread by coughing, sneezing, loud talking or laughing. The recovery of the virus from sewage and flies in infected areas makes one believe that there are indirect methods of spread.

In 1933 it was still thought that the olfactory tract was a common portal of entry of the virus and we all sprayed our noses with zinc sulphate until we lost our sense of smell for months. Since then numerous examinations of the olfactory bulbs of patients who have died of poliomyelitis have failed to demonstrate, except in a few instances, the virus or its inflammatory reactions in the bulb.

Experimental work on the cynomalgus monkey and chimpanzee with oral, gastric, and rectal virus feeding has been carried out by Sabin, Ward, Toomey, Howe, Kling, Faber and others. Some believe the route of penetration is through the sympathetic nerve fibers of the gut to the celiac plexus and thence to the spinal cord. Flexner in 1936 believed entry was through the nasal and buccal mucous membranes, and not the intestine, and the presence of the virus in the stool was excretory rather than invasive. In 1936 Toomey first suggested a very plausible route of invasion through the vagus nerve or the afferent fibers of the nodose ganglion which supplies both the upper and lower alimentary tract and is a very possible route because of the wide distribution of the nerve fibers.

Very recent experimental work of Faber, Silverberg and Dong points to peripheral ganglia, especially the gasserian, as the most often involved. This supports the theory of the primary portal of entry being the upper alimentary tract (the mouth, the pharynx and perhaps the esophagus).

Therefore, the consensus of opinion regarding the portal of entry at the present time is that the virus enters through the mucous membrane of the alimentary tract, upper, lower, or both.

Having once entered a susceptible person the organism may multiply rapidly and invade the brain and the spinal cord and possibly the blood stream. The inflammation of the nerve cells may cause weakness and paralysis of certain muscles; these may recover if the nerve cell is not destroyed. The pathologic changes of the nerve cells are found in the anterior horn cells, basal and posterior ganglia cells.

It is important, at least in the four months of epidemic years when poliomyelitis most often occurs to follow the suggestions of the Kansas State Board of Health and the Chicago and Boston plans and adopt certain measures to protect the individual against receiving a dose of virus large enough to cause the serious complication of poliomyelitis.

1.—Unnecessary meetings should be omitted and one should avoid congested places.

2.—Personal contact, such as kissing and hand shaking, should be avoided.

3.—Pets should not be handled and steps should be taken for protection against insects of all kinds.

4.—People should avoid using drinking and eating utensils outside of their own home.

5.—Milk should be pasteurized or boiled.

6.—Individuals should keep fit, eat proper foods, and get plenty of rest and fresh air.

7.—Use of nasal antiseptics is not advised as it disturbs the normal protective mucous film on the mucous membranes of the nose.

8.—Swimming should be avoided as water may be contaminated.

9.—Operations on the nose and throat, especially tonsils and adenoids, should be postponed.

In nonepidemic years it would be wise to follow the first eight of these suggestions as a postoperative guide to prevent exposure to the virus in the summer months.

CONCLUSIONS

1.—The possibility of poliomyelitis during July, August, September and October in nonepidemic years following tonsillectomy and adenoidectomy is minimal, but when it does occur, it is serious and most often bulbar in type. Ratio: one case in 2,000 operations.

2.—The possibility of poliomyelitis following tonsillectomy and adenoidectomy in the other eight months is nil, except in Texas and California where the poliomyelitis season is extended from June to November and sometimes includes December.

3.—Preventive medicine as practiced now by Boards of Health throughout the nation shows that during months and years when

there are epidemics of poliomyelitis operations on the nose and throat should be avoided whenever possible for the safety of patients, and I believe it is wise for otolaryngologists to cooperate in this respect.

FOURTH AND VINE STREETS.

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TABLE 1

POLIOMYELITIS FOLLOWING RECENT TONSILLECTOMY ACCORDING TO CLINICAL REPORTS

Type of Disease Interval: Within 30 Days					Within 30 to 60 Days					Total Cases	References
B	BS	Unkn.	S	NP	B	BS	Unkn.	S	NP		
1										1	Sheppard, 1910.
				1						1	Boyd, 1912.
9										9	Ayer, 1928.
7	5		4							16	Aycock & Luther, 1929.
4		2								6	Gordon, 1931.
5										5	Silverman, 1931.
4			2	3						9	Ontario Health Dept., 1937.
2										2	Anderson, 1938.
15	2		4	3	1			3	1	29	Elly & Flake, 1938.
6	3		2	2						13	Stillerman & Fischer, 1938.
1										1	Pamment, 1933.
1				2						3	Krammer & Gilliams, 1938.
1										1	Stebbins, Gillick & Ingraham, 1939.
2										2	Koskoff, 1939.

TABLE 1—(Continued)

POLIOMYELITIS FOLLOWING RECENT TONSILLECTOMY ACCORDING TO CLINICAL REPORTS

Type of Disease Interval: Within 30 Days					Within 30 to 60 Days					Total Cases	References
B	BS	Unkn.	S	NP	B	BS	Unkn.	S	NP		
5	2		2	3	2			13		27	Fischer, Stillerman & Marks, 1941.
2	3		1	1						7	Helms, 1941.
5										5	Krill & Toomey, 1941.
15	5	5	5	3	5		4	11		53	Vt., 1912-31, Mass., 1927.
14	2		2	2		1	1	3		25	Personal Communications.
3			3							6	Howard (Cincinnati, 1937-43).
4			2		3			3		12	Kinnaman (Kansas, 1940 to 1943).
3			1							4	Wynns (California, 1943).
8	2		2		1	1			1	15	Piszczek (Cook County) 1943. Walsh (Du Page County) 1943.
	1		4		—				2	7	McFarland (Los Angeles) 1943.
117	25	7	34	20	12	2	5	33	4	259	

B—Bulbar
S—Spinal
BS—Bulbo-spinal
NP—Nonparalytic
Unkn—Type Unknown

Adapted from Aycock, 1942.

TABLE 2

DATA ON CHILDREN IN WHOM POLIOMYELITIS DEVELOPED WITHIN 30 TO 60 DAYS AFTER REMOVAL OF TONSILS AND ADENOIDS

CASE	PATIENT	AGE	DATE OF OPERATION	DATE OF ONSET POLIOMYELITIS	INTERVAL DAYS	TYPE OF POLIO
Cook County 1943 (Epidemic Year)						
1	C. L.	9	9-17-43	8- 4-43	18	B (Died 8-8-43)
2	Case # 53					
	L. D.	5	7-31-43	8-15-43	16	B
3	M. H.	7	7-17-43	7-27-43	10	BS
4	M. H.	3	8- 5-43	9- 5-43	30	S
5	K. G.	8	7-23-43	8-15-43	23	B (Died 8-19-43)
6	D. H.	6	7-30-43	9-28-43	59	P
7	Case # 11	3	7-20-43	8- 2-43	13	B (Died 8-8-43)
8	Case # 57	4	7-23-43	8-17-43	25	BS
9	Case # 62	8	7-31-43	8-15-43	16	B (Died 8-19-43)
10	Case # 80	6	7-16-43	7-27-43	11	B
Du Page County 1943 (Epidemic Year)		41 Cases of Poliomyelitis—9 Bulbar—6 Deaths.				
1	K. F.	8	8- 1-43	9-14-43	45	B (Died 9-18-43)
2	A. F.	5	8- 1-43	9-27-43	58	BS
3	M. L.	11	8-21-43 Tonsil Tag	9 -3-43	13	B (Died 9-7-43)
4	M. W.	5	8-26-43	9-10-43	15	B (Died 9-11-43)
5	M.	4	10-1-43	10-10-43	10	S

Cases Reported from Cook and Du Page Counties through the courtesy of Drs. Piszczek, Walsh and Levinson.

Fifteen cases reported this year in the Chicago area following recent tonsil and adenoid removal show the majority occurred in patients operated on in July. These figures speak well for preventive medicine as practiced by Chicago and Cook County Public Health Staffs.

Kansas and California also have had epidemic years with post-tonsillectomy poliomyelitis cases as reported by their epidemiologists.

TABLE 2—(Continued)

DATA ON CHILDREN IN WHOM POLIOMYELITIS DEVELOPED WITHIN 30 TO 60 DAYS AFTER REMOVAL OF TONSILS AND ADENOIDS

CASE	PATIENT	AGE	DATE OF OPERATION	DATE OF ONSET POLIOMYELITIS	INTERVAL DAYS	TYPE OF POLIO
Cases reported by Dr. C. H. Kinnamon, Kansas State Epidemiologist.						
1	J. B.	6	7-4-40	8-4-40	30	B
2	D. H.	8	8-15-40	9-4-40	19	B
3	W. C.	9	8-24-42	9-9-42	16	B (Died)
4	J. W.	4	7-10-43	8-14-43	35	B (Died)
5	M. M.	9	7-17-43	8-7-43	21	B
6	V. R.	6	7-19-43	8-4-43	16	S
7	B. S.	7	8-2-43	8-14-43	12	S
8	N. T.	12	6-39-43	8-28-43	60	S
9	L. W.	7	6-3-43	8-3-43	60	S
10	G. S.	9	6-20-43	8-13-43	54	B
11	C. R.	8	7-1-43	9-1-43	60	B
12	G. C.	6	7-13-43	9-8-43	57	S

TABLE 2—(Continued)

DATA ON CHILDREN IN WHOM POLIOMYELITIS DEVELOPED WITHIN 30 TO 60 DAYS AFTER REMOVAL OF TONSILS AND ADENOIDS

CASE	PATIENT	AGE	DATE OF OPERATION	DATE OF ONSET POLIOMYELITIS	INTERVAL DAYS	TYPE OF POLIO
Cases reported by Dr. H. L. Wynns, California State Epidemiologist and Dr. W. McFarland, Los Angeles, Calif.						
	Case # 1	4	7-6-43	7-17-43	11	B (Died)
	Case # 2	6	7-6-43	7-20-43	14	B (Died)
	Case # 3	5	7-4-43	7-25-43	21	S
	Case # 4	6	7-7-43	7-28-43	21	B (Died)
	Case # 5	6	8-11-42	8-24-42	14	B (Died)
	Case # 6	6	8-13-42	8-27-42	14	S
	Case # 7	5	8-1-42	10-1-42	60	S
	Case # 8	9	9-16-42	10-6-42	31	S
	Case # 9	8	10-7-42	10-14-42	7	S
	Case # 10	4	11-4-42	11-22-42	18	S
	Case # 11	6	6-18-43	8-18-43	60	S

B—Bulbar S—Spinal BS—Bulbo-Spinal

TABLE 3.—INCIDENCE OF POLIOMYELITIS AFTER TONSILLECTOMY AND ADENOIDECTOMY IN CINCINNATI HOSPITALS.

YEAR	NO. POLIOMYELITIS CASES	POLIOMYELITIS CASES JULY, AUG., SEPT., OCT.	TOTAL TONSILLECTOMIES	TONSILLECTOMIES AND ADENOIDECTOMIES	TONSILLECTOMIES AND ADENOIDECTOMIES JULY, AUG. SEPT., OCT.	POLIOMYELITIS RECENT TONSILLLECTOMY AND ADENOIDECTOMY BULBAR	SPINAL
1937	93	92	4608	2842	1580	1 7-23-37	1 8-2-37
1938	2	2	4839	3004	1754	0	0
1939	14	14	5005	3756	1681	0	2 7-29-39 8-1-39
1940	68	60	4963	3069	1728	0	0
1941	26	20	5140	3174	1872	0	0
1942	34	27	5863	3789	1965	1 7-12-42	0
1943	20	18	5877	3808	2380	1 9-1-43	—
	257	233	36,295	23,442	12,960	3	3

Statistics received through cooperation of the Cincinnati Board of Health and the following hospitals: City, Christ, Good Samaritan, Jewish, Childrens, Deaconness, Holmes, and Bethesda.

TABLE 4.—DATA ON CHILDREN IN WHOM POLIOMYELITIS DEVELOPED WITHIN 30 DAYS AFTER REMOVAL OF TONSILS AND ADENOIDS (CINCINNATI).

CASE	PATIENT	AGE	DATE OF OPERATION	DATE OF ONSET POLIO	INTERVAL DAYS	TYPE OF POLIO
1	N. Z.	5	6-15-37	7-2-37	17	BS
2	P. M.	3	7-12-37	8-2-37	21	S
3	L. U.	8	7-15-39	8-1-39	17	S
4	E. U.	4	7-15-39	7-29-39	14	S
5	R. S.	4	6-28-42	7-12-42	14	B
6	R. M.	6	8-24-43	9-3-43	10	B (Died 9-4-43)

B—Bulbar S—Spinal BS—Bulbo-Spinal

TABLE 5.—INCIDENCE OF POLIOMYELITIS IN THE UNITED STATES IN 1943.

	JAN.	FEB.	MAR.	APRIL	MAY	JUNE	JULY	AUG.	SEPT.	OCT.	NOV.	DEC.
NEW ENGLAND												
Maine, New Hampshire, Ver., Mass., R. I. and Conn.	11	0	63	3	1	9	21	195	314	217	59	13
MIDDLE ATLANTIC												
New York, New Jersey Penn.	7	8	11	6	11	13	50	161	329	262	72	30
EAST CENTRAL												
Ohio, Ind., Ill., Mich., Wis. (Illinois epidemic second week of Aug. and Sept.)	14	9	9	7	15	5	46	510	1017	594	134	37
WEST												
Minn., Iowa, Neb., Kan., Missouri, N. & S. Dakota (Kansas epidemic Aug. and Sept.)	14	12	5	5	5	9	80	427	580	309	49	16
SOUTH ATLANTIC												
Delaware, Maryland, D. of C., West Va., Va., N. & S. Carolina, Ga., Fla.	12	16	10	6	5	13	24	31	47	43	15	5

TABLE 5.—(Continued)

	JAN.	FEB.	MAR.	APRIL	MAY	JUNE	JULY	AUG.	SEPT.	OCT.	NOV.	DEC.
EAST SOUTH CENTRAL												
Ky., Tenn., Ala., Miss.	10	6	7	14	12	7	25	65	39	28	20	4
WEST CENTRAL												
Ark., Louisiana Okla., Tex. (Texas epidemic in July)	29	11	17	7	26	108	687	462	327	158	91	29
MOUNTAIN	7	11	8	14	16	13	36	141	395	203	69	27
PACIFIC												
Wash., Ore., Calif. (California epidemic)	32	21	18	22	55	168	451	625	641	660	338	76

Computed from statistics received from the National Foundation for Infantile Paralysis.

TABLE 6.—POLIOMYELITIS IN STATES WHERE EPIDEMICS OCCURRED—1937 TO 1943.

STATE	YEAR	JUNE	JULY	AUG.	SEPT.	OCT.	NOV.	DEC.
ILLINOIS	1937	2	49	160	383	151	28	8
	1938	6	12	31	22	13	3	5
	1939	6	20	36	64	27	16	2
	1940	7	6	59	224	150	122	19
	1941	10	23	93	102	71	58	17
	1942	8	24	111	182	122	45	7
	1943	3	20	415	729	461	83	13
KANSAS	1937	2	20	54	100	63	6	2
	1938	0	1	2	0	1	0	0
	1939	2	3	14	14	9	6	3
	1940	7	24	164	205	94	34	4
	1941	0	0	6	17	11	6	3
	1942	2	3	9	30	43	9	10
	1943	2	52	274	266	129	34	5

TABLE 6.—(Continued)

STATE	YEAR	JUNE	JULY	AUG.	SEPT.	OCT.	NOV.	DEC.
TEXAS	1937	9	184	188	107	92	16	18
	1938	3	6	9	6	5	3	5
	1939	11	45	36	55	36	21	12
	1940	8	26	49	16	20	11	3
	1941	7	18	18	15	22	13	8
	1942	7	6	16	11	48	53	90
	1943	84	473	256	210	98	42	16
CALIFORNIA	1937	25	79	138	156	103	61	17
	1938	6	22	20	12	14	2	8
	1939	48	166	713	227	142	92	54
	1940	86	74	81	57	45	19	11
	1941	25	21	42	35	24	28	8
	1942	5	9	29	36	81	51	51
	1943	165	437	506	492	365	204	43

III

THE PHYSIOLOGY OF DRAINAGE OF NASAL MUCUS
IV. DRAINAGE OF THE ACCESSORY SINUSES IN MAN

Rationale of Irrigation of the Infected Maxillary Sinuses

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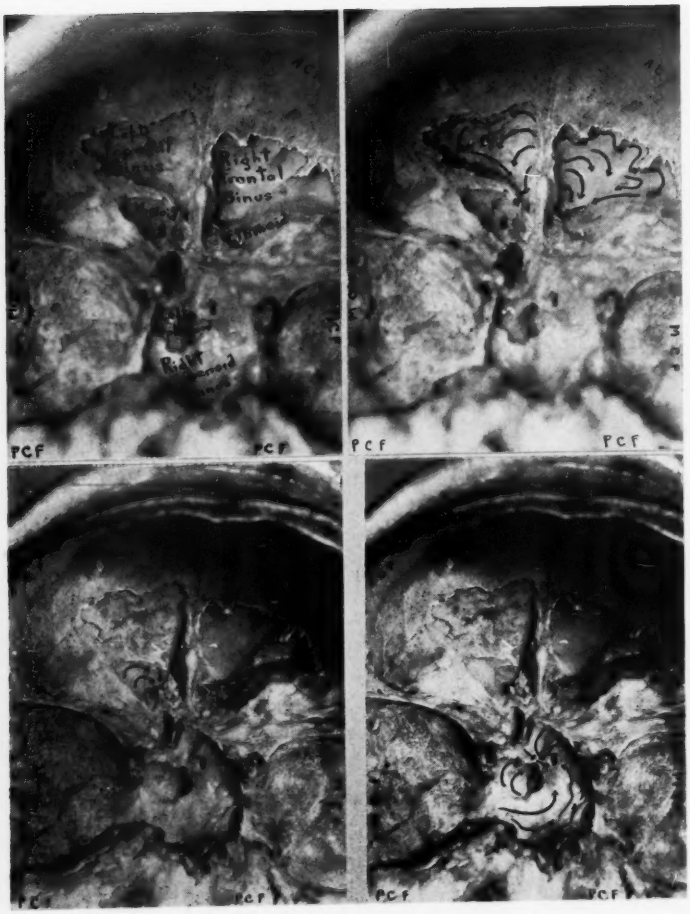
It had been hoped in this fourth article of the series to give a complete study establishing the manner of the drainage of mucin from all of the nasal sinuses in man. It has been so difficult to obtain sufficient suitable material that the study is by no means complete. The data obtained, although fragmentary, seem consistent with the findings in experimental animals[1] and are probably worthy of a brief report.

Necropsy material* was used in studying all but the maxillary sinus. It was necessary to attend routine necropsies rather faithfully for several months to find the occasional case in which the ciliary action could be well observed post mortem. Two requirements that seemed to coincide surprisingly seldom were essential: permission to open the cranium and a body sufficiently warm so that the ciliary action was still largely unimpaired. Several such subjects were eventually obtained. After the brain had been removed, the sinuses were exposed in the base of the cranium. Small drops of India ink, placed in various portions of the sinuses, were used as indicators of the direction of flow. The time during which active flow lasted after the head was opened and the brain removed was rather brief in each case—never as long as desired. This was unexpected because microscopic bits will remain active under the microscope for hours. In some instances the ciliary action was stimulated by flushing the sinuses with a warm, mildly alkaline solution of normal saline.

The study reported here was made a number of years ago, and has been discussed at various meetings, but has never been published.

*Work done at St. Luke's Hospital, Duluth.


Right
frontal
sinus
PCF
PCF
PCF
PCF
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PCF

Fig. 1.—Photograph of the base of the skull as seen from the cranial cavity. Both frontal sinuses, both sphenoid sinuses, and one ethmoid sinus on each side have been unroofed. The initials refer to cranial fossae: ACF and MCF and PCF indicate anterior, middle, and posterior fossae respectively.

This photographic view is used in the other four illustrations.

Fig. 2.—Plan of ciliary streaming in the frontal sinus as indicated by the movements of dots of India ink placed upon the mucous membrane. In general, the direction is a spiral that flows laterally and posteriorly.

Fig. 3.—Plan of ciliary streaming in two ethmoid sinuses as indicated by the movements of dots of India ink placed upon the mucous membrane.

Fig. 4.—Plan of ciliary streaming in the sphenoid sinuses, as indicated by the movements of dots of India ink placed upon the mucous membrane.

In this subject the right sphenoid was found to be very large, whereas the left was small.

Observations on the maxillary sinuses were made by means of a nasopharyngoscope through the operative window following modified Caldwell-Luc operations. The mucous membrane had not been removed from these sinuses at the time of operation. Thirty-eight sinuses were examined. The method is limited because all surfaces could not be seen, but the lines of flow could be well observed on such surfaces as could be brought into view through the instrument. These were: part of the floor, the lateral and anterior walls, much of the ceiling, and a portion of the medial wall above and below and posterior to the window. The ostium could be seen in many. In many no data were obtained. In some, small bits of mucin stretched along the walls toward the ostium indicated the lines of flow. The best observations were obtained in four patients, who, good naturedly, permitted a drop or two of India ink to be placed on the floor.

RESULTS

In general, each sinus observed exhibited a spiral manner of drainage similar to that in the frontal sinus of the dog. The direction of the spiral in the frontal sinus is illustrated in Fig. 2. No very satisfactory observations were made in the ethmoids. The best one showed a spiral that was directed anteriorly and inferiorly across the lateral wall and thence medially across the anterior wall and floor to the ostium, (Fig. 3).

Two sphenoids observed in one subject exhibited parallel instead of symmetrical spirals about the ostia; that is, the direction of the whirl at the ostium as it crossed above the opening was toward the left in both sinuses. In the left sinus this was lateral in direction, and in the right it was medial. The spiral, as found in the right, is shown in Fig. 4. In this sinus, the spiral was very similar to the one found regularly in the frontal sinus of the dog[1]; that is, from the deeper portions of the cavity, laterally up and anteriorly across the lateral wall, then medially across the anterior wall to the ostium.

The ink placed on the floor of the maxillary sinuses in each case spiraled somewhat laterally and then upward to the anterolateral wall, across this anteromedially, and upward to the ostium. Some crossed the roof just before reaching the ostium. In none was the ink observed to pass out through the artificial window. In fact, in one instance a film of clear mucin containing streaks of ink was seen to span across the lower portion of the window without getting out into the inferior meatus. Apparently this mucin was on its

Fig. 5.—Movements of dots of India ink placed upon the mucous membrane in a sphenoid sinus which contained an abnormally large quantity of secretion. The subject was lying upon his back. The dots moved in an irregular spiral toward the ostium until the climb became too steep, and then, carried by the secretion, slid back to the bottom of the cavity, as indicated by the dotted lines. Some actually crossed above the ostium and slid down again on the other side.

way to the ostium and was dragged by traction across the lower part of the window. I do not mean to imply that the operative window is useless. Obviously, if a large mass of secretion were present, it would flow out through the window by gravity into the inferior meatus and, by ciliary action, be carried posteriorly into the pharynx. In those sinuses containing comparatively small amounts of abnormal secretion, this also spiraled upward and toward the ostium and did not pass out through the window.

Rationale of Sinus Irrigation

One of the sphenoids examined held a small amount of creamy, purulent secretion containing very little mucin. The movements of this secretion, as indicated by the ink droplets, were very illuminating as to the manner in which abnormal secretion can accumulate when the ostium lies high above the floor of a sinus (Fig. 5).

This brings up the question of the rationale of sinus irrigation. Keeping in mind the physiology of ciliary drainage, what do we accomplish by irrigation of the maxillary during an attack of sinusitis? That depends upon the nature of the pathological conditions present. If the sinus is filled with creamy pus and the mucous membrane entirely destroyed, then the condition is essentially an abscess cavity in a bony tissue, and removal of the pus has the same beneficial effects that it has in other abscesses. Usually, however, the lining mucous membrane is not entirely destroyed, and the condition is somewhat different from that in the case of an abscess cavity. The exudation is not in direct contact with the connective tissues, but is separated from them by a surface of mucous membrane as far as this remains intact. By means of its ciliary activity, this mucous membrane drains the sinus of the normal thin film of mucin about every 15 minutes. Gravity does not materially retard the drainage of the normal film as it passes up the more or less vertical walls to the ostium, because it is thin enough, and viscid enough, to adhere closely to the tips of the moving cilia. When the amount of the secretion is sufficiently increased, however, the film becomes so deep that when the vertical walls are reached, only the deepest portions of the film next to the tips of the cilia go on through the ostium, while the superficial portions of it slide back to the bottom of the cavity by gravity. In this manner, the secretion may accumulate until the cavity is full. This was very well illustrated by the sphenoid pictured in Fig. 5. The chances of this accumulation forming are further enhanced if the content of mucin in the secretion is decreased because it slides back more easily. (The cilia handle a heavy mucinous secretion better than other types.) The volume need not become very great before the cilia are many hours behind in their duty of keeping the cavity empty. If the accumulation of secretion can be removed by irrigation, the deleterious effects, whatever they may be, are avoided, and the cilia are relieved of a great many hours of labor and given a fresh start. If they are not given this help, the destruction may become sufficiently great to allow the infection to become chronically established.

SUMMARY

All of the sinuses of man seem to drain the normal film of mucin with a spiral motion centering at the ostium. The direction of the flow of mucin in the maxillary sinus is not altered by making a window into the inferior meatus. It is still directed toward the ostium as it was before. When an abnormally large quantity of secretion is contained, this also is directed toward the ostium al-

though much of it doubtless flows out of the window by means of gravity.

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IV

TERMINAL STAGES IN THE DEVELOPMENT OF THE HUMAN STAPES

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In the course of its morphogenesis as a cartilaginous, and later as an osseous, skeletal element in vertebrates, the stapes has undergone a remarkable transformation. Some of the more primitive steps in the alteration are repeated in the individual developmental history of the ossicle in man, while others are not only peculiar to the stapes but also unique in the general history of bone formation. The predecessor of the stapes functioned as a support for the respiratory mechanism in aquatic and amphibious vertebrates; in mammals it has been made over to serve as the terminal link in an ossicular chain whose function it is to convert air waves into liquid waves. In response to these new demands, the stapes of the human embryo, appearing first as part of the branchial arch system, later establishes anatomic relationships at one extremity with another ossicle, at the other with an orifice in the capsular shelter for the acoustic end-organs. The stapes develops rapidly, concomitant with a vestibular window whose dimensions are relatively fixed near the middle of fetal life, growth thereupon becoming minimal. The ossicle adapts its histologic structure to an existence within a chamber which is early invaded by pharyngeal mucous membrane, and concurrently sacrifices considerable bulk to reduce inertia. These anatomic objectives and the developmental means employed to attain them will be the subject of the current article.

The observations were made chiefly upon series in the collection of the Department of Anatomy of the University of Wisconsin;

From the Department of Anatomy and the Department of Otolaryngology, Northwestern University Medical School (contribution No. 407 from the former).

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these were made available for study through the courtesy of Professor Theodore H. Bast. The drawings were made from wax-plate reconstructions.*

As pointed out in earlier articles,[3, 5] the human stapes makes its appearance as a concentration of mesenchymal cells in the 7-mm. (4-week) embryo, the stapedial blastema arising from the cranial extremity of the second branchial or hyoid bar. This hyostapedial connection is represented in later stages not by a cartilaginous element, but by the stapedius muscle and tendon. At the 25-mm (8 week) stage the stapes has lost its connection with the branchial skeleton but has established a new articular association laterally with the incus, and medially with tissue of the vestibular (oval) window—which will later contribute to the substance of the annular ligament. Thus, beginning as a branchial element, the stapes becomes intimately associated with the otic capsule.

In the 30-mm. (8½-week) embryo the stapedial cartilage is still a simple ring. But having established relations with permanent structures, it undergoes rapid change in form; and in the 50-mm. (11-week) fetus the definitive portions of the stapes are clearly recognized: a short cylindrical head and a flattened base are set off from the diverging crura. In the 75-mm. (13-week) specimen, it is truly "stapedial" in form, but still entirely cartilaginous in fabric.

The stapes of the 100-mm. (15½-week) fetus is a cartilaginous structure of true stirrup form (Fig. 1),** modified from the original ring-shaped element of smaller size. A cylindrical head and flattened base join equally robust crura; the intercrural space, or obturator foramen, is relatively small and roughly circular. The outlines of the stapes as a whole are evenly contoured, with no definite

*The wax-plate reconstructions were prepared at a magnification of 125 diameters; the figures in Fig. 7 were drawn without reduction, those in Figs. 1-4 at one-half original size; all were considerably reduced in reproduction. For the sake of simplicity in labelling, no distinction has been made between the head and the neck of the "capital" portion of the stapes.

**The stapes of the 100-mm., 150-mm. and 183-mm. fetuses, and of the 57-year adult have been more fully described in earlier accounts emanating from this laboratory. In articles published in the Archives of Otolaryngology certain developmental features have been described in detail. Two initial articles[1, 2] accounted for selected crucial stages from the 7-week embryo to the 70-year adult; two succeeding articles[3, 4] and others now in preparation complete the description of the stapes, and fissula ante fenestram, in numerous graded series from the 6.7-mm. embryo to the adult of advanced age.

Other general features of development and of gross form of the stapes have been presented in the Quarterly Bulletin of the Northwestern University Medical School[6-8].

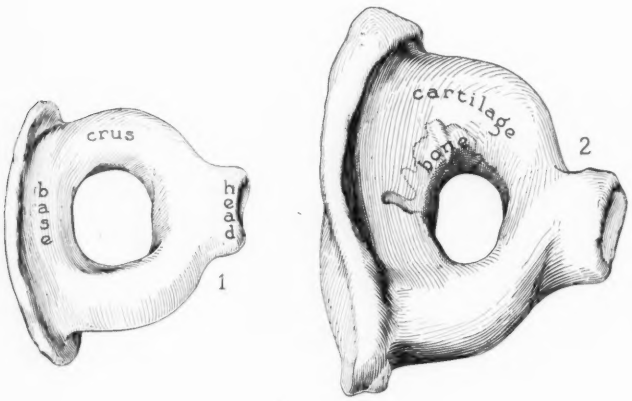

Fig. 1.—Stapes of 100-mm. fetus (reconstruction, entire). x18.

Fig. 2.—Stapes of 150-mm. fetus (reconstruction, entire). x18.

division between either head and crura or between crura and base. All portions are composed of solid unaltered cartilage of hyaline type and invested by an active perichondrium.

Although the stapes of the 150-mm. (20-week) fetus is still destined to undergo profound alteration in histologic structure and in some features of gross form, yet it already possesses several morphologic characters which it will retain throughout the life of the individual (Fig. 2): the base is lipped marginally and is reniform in outline; the vestibular surface is undulate, the head is foveate for articulation with the incus; and the crura are unequal in size and different from each other in form, the posterior being the bulkier and more bowed of the pair.

The assumption of stapedial configuration, at this stage, is concurrent with the initiation of bone formation. Once ossification begins, the ensuing changes are those of remodelling; and although the chondral tissue of the original cartilaginous model is largely replaced by bone in the ordinary way, the latter tissue is modified by a series of extraordinary changes. This succession of steps is introduced, in the stapes of the 150-mm. fetus, by the development of a center of ossification on the tympanic (lateral) surface of the base. Periosteal bone appears on the summit of the basal part, spreads rapidly, to descend on both aspects (superior and inferior) of the base and to

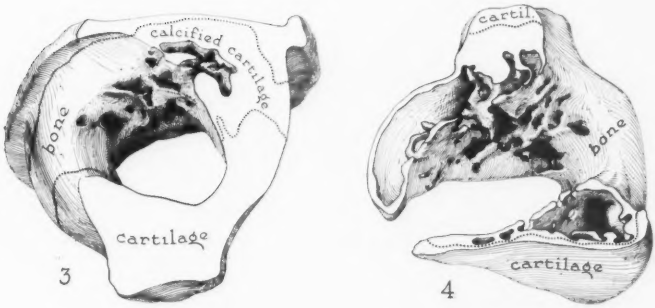

Fig. 3.—Stapes of 160-mm. fetus (reconstruction, partial). x18.

Fig. 4.—Stapes of 180-mm. fetus (reconstruction, partial). x18. In this figure and in Fig. 3 an upper piece has been removed in the plane of section to display the interior structure. Cut surfaces are plain white.

ascend toward the head on the obturator surface of each crus. Further bone formation is accomplished through similar spread, since there is but one center of ossification for the entire ossicle. Just preceding this phase, the cartilage of the area becomes rarefied and gradually undergoes calcification. A very thin sheet of periosteal bone then covers the rarefied cartilage, in a manner similar to that by which the cartilage model of any long bone (e.g., a tibia) is converted into an osseous tube.

The next crucial stage in stapedial morphogenesis is that observed in the 160-mm. (21-week) fetus. The shell of bone now surrounds each crus and descends to the level of the lipped margin of the base (Fig. 3); but it has not yet included the head of the stapes. The cartilage newly invested by the periosteal shell is deeply eroded and calcified.

In the 180-mm. (23-week) fetus, ossification has extended to the crural end of the head so that periosteal bone now forms a complete tube (Fig. 4). It is continuous around the entire internal or obturator surface, incomplete on the external aspect where it meets the cartilage of the base and that of the head. At this stage the cartilaginous head is but slightly eroded, whereas the base is so markedly excavated that cartilage remains only as a vestibular plate; the crura are completely freed of cartilage core. The tubular bony crura

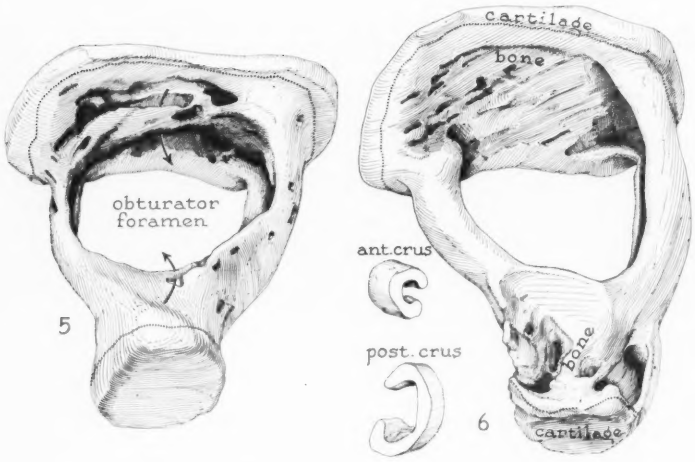

Fig. 5.—Stapes of 210-mm. fetus (reconstruction, entire). x18.

Fig. 6.—Stapes of term fetus (reconstruction, entire). x18.

are foraminous on the obturator surface. These foramina are larger, more numerous, and more widely distributed than in the preceding stage of development. They are present on the corresponding aspects of the head and base. As in any fetal long bone, they serve to transmit osteogenic buds of vascular connective tissue into the spaces covered by the periosteal shell; but they are almost wholly confined to the obturator surface of each of the several parts of the stapes. The progress of development in the capital end is slower than either in the crura or in the base—a lag in morphogenesis which remains characteristic of more advanced stages. Whereas cartilage, partially converted into intrachondral (calcified cartilage) and endochondral bone, remains for some time in considerable amount in the head, such tissues persist in the base chiefly as a plate on the vestibular surface.

The ensuing stages in structural modification of the stapes are even more strikingly divergent from those which characterize the maturation of a regular long bone. The outcome of these novel steps in morphogenesis are evidenced in the fetus of 210-mm.

In the 210-mm. (25-week) fetus erosion results in almost complete removal of periosteal bone from the internal or obturator aspect

of the stapes (Fig. 5). At some points the free margin is still irregular or crenated where originally separate foramina have coalesced but have not yet been smoothed marginally. The crura now resemble channelled beams; they contain but few spicules of endochondral bone; their primitive marrow is being rapidly destroyed to make way for tissue of submucosal character. The head still contains irregular hummocks of calcified cartilage whose free ends are composed of endochondral bone. The base is not quite as simple in structure, since some of the endochondral bone, which covers the basal cartilaginous plate and occasionally a portion of the overarching roof of periosteal bone, persists along the margin and across the base between the crural attachments. In some specimens, only the peripheral ledge of the persisting bone turns inward to fuse with the subjacent endochondral plate, thus increasing the thickness of the base around the circumferential lip; in others, the median stripe also remains, apparently to form the inconstant crista of the adult ossicle. In the 210-mm. stage fetal stapes the crista and the osseous base are connected by very irregular masses of osseous tissue. Assumedly, through the operation of slow processes of remodelling, the several portions merge to produce the less conspicuous crest of the older ossicle.

In the 245-mm. (29-week) fetus the stapes shows progress along each of the developmental lines discussed above. On the base the marginal ledge of periosteal bone has fused with the endochrondral bone covering the lamella of cartilage, enveloping blood vessels in primitive haversian-like spaces; the flattened field of the central area rises to a thicker peripheral part and is crossed, between the crura, by an inconspicuous and incomplete crest. In the head endochondral bone has been largely removed; the crura are further eroded and are consequently more widely open on the obturator aspect. This process is not equal in the two crura; in this specimen, and commonly in others, the anterior crus is made the slenderer and thinner of the two. Except for the persistence of endochondral bone in the head of the stapes, the ossicle of the 245-mm. fetus could be mistaken for an adult specimen.

Adult configuration and histologic structure have been attained by the stapes of the 275-mm. (31½-week) fetus. Like the ossicle of the term fetus (Fig. 6), the head and base are bilaminar; cartilage persists only as a local covering for the osseous plates. Mucous membrane has invaded the capital, basal and crural portions, replacing the original marrow. In some postnatal specimens of stapes the head is eroded externally, so that it is either deeply pitted or nar-

rowed, or its upper and lower walls are removed unequally. On theoretical grounds, it would be expected that such erosion should constitute a phase of the osteoporotic changes associated with old age. Actually, as here demonstrated, they are established in the fetus. Other skeletal elements show a constancy in form and development dependent upon the forces and functions which they subserve. It is remarkable that the stapes, so early subjected to forces of irregular erosion, should maintain such regularity in the form of its main features.

The succession of changes which occur in each of the three major portions of the ossicle may now be considered.

Of the three portions of the stapes, the crus follows the shortest series of developmental steps (figures in first column of Fig. 7). A cartilaginous column (100-mm. fetus) is converted into an osseous tube (160-mm. and 183-mm.) by a mechanism generally similar to that which takes place in the formation of the shaft of any long bone, but different therefrom in the concentration of vascular orifices on one surface. Next, and departing further from the familiar plan of ossification, the obturator surface of each crus, rendered foraminous (183-mm.), is completely resorbed through indenting and coalescence of the originally separate foramina (term fetus). Mucous membrane (not shown in the figure) invades the channelled member, replacing marrow and the small amount of persisting endochondral bone. The periosteum ceases to be productive as soon as bone forms a complete crural tube, and hence the crura do not enlarge perceptibly after midterm. A crus comes to resemble a miniature long bone halved longitudinally, robbed of its marrow and cancellous tissue and invested completely by mucous membrane.

The head is similarly eroded from the obturator aspect, but the removal of cartilage is a slower process (figures in the second column of Fig. 7). The capital part of the stapes is a cylinder closed at its lateral, or articular, end; excavation therefore takes place on the opposite or obturator extremity. Cartilage (100-mm.) is eroded (183-mm.), and endochondral bone is formed in relative abundance, through the conversion of calcified cartilage (180-mm. and 245-mm.). The tips of the cartilage hummocks are first ossified. These islets of bone spread, and concurrently with destruction of cartilage upon which they rest, fuse to form a lamella of endochondral bone mergent with the periosteal layer of the capital portion. Most of the irregular, "cancellous" tissue is removed by this process to make a hollow head whose walls are usually smoothed. In some specimens,

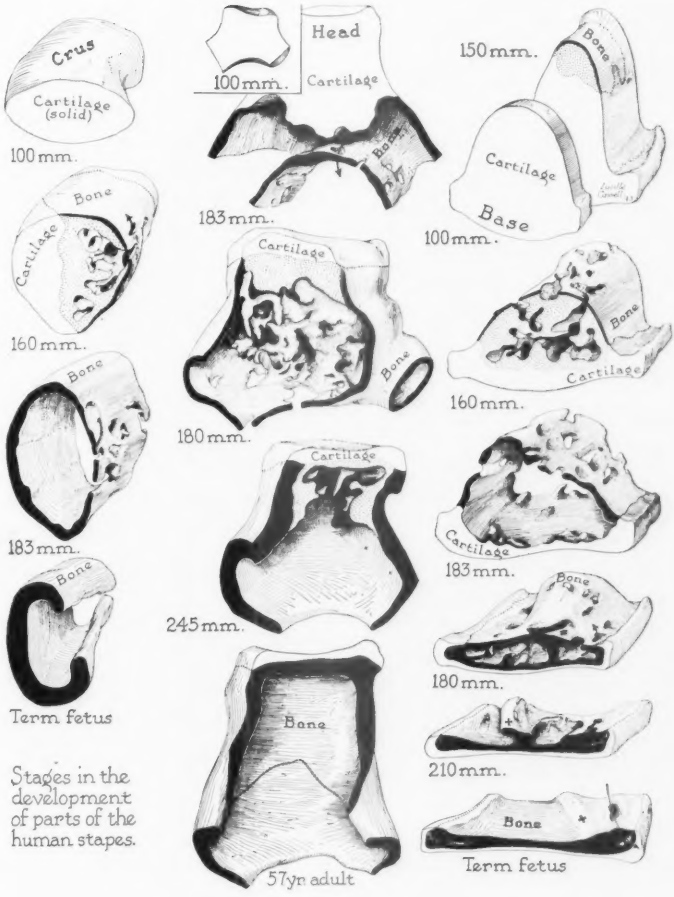

Fig. 7.—Portions of stapes in developmental succession: left line, crural portion; middle, capital; right, basal. All figures semidiagrammatic, from actual reconstructions. x31. Crural segments were taken near capital end of posterior crus; capital segments, in longitudinal section to include neck and crura; basal, at portion near posterior crus. On cut surfaces cartilage is shown plain (white); bone, black; calcified cartilage, stippled. On natural and on cut surfaces, dotted line indicates junction of cartilage and bone, except in lower middle figure where it marks outline of superior wall of head (removed). Arrows pass through foramina. Crosses indicate basal crest.

however, a portion of endochondral bone may persist in the form of a ledge which encroaches upon the space of the head (245-mm.). Marrow is replaced as the mucous membrane of the tympanic cavity invades the head, just as it is in the crura. Cartilage persists only on the articular surface, where it is lined by a thin lamella of endochondral bone—to make that articular surface bilaminar (adult). No epiphysis occurs; therefore, the head does not lengthen after it has been formed in bone. The periosteum, like that of the crura, remains virtually inactive; consequently the head does not widen. Thus, the head of the ossicle differs from the extremity of a typical long bone in lacking epiphysis, as well as cancellous core and marrow, and in being covered on the exterior and the interior by mucous membrane; it is similar to such a part in being articular and cylindrical.

Developmental steps at the basal extremity make up an even more complex succession of patterns (third column of Fig. 7). The ossification center which ultimately spreads from the base along the crura to the head, forming a tubular ring of bone, first appears on the obturator surface of the base (150-mm.; cf. 100-mm.). On this surface profound structural changes occur, while the opposite, or vestibular, surface and the circumferential area for fenestral articulation remains simple cartilage. As in the case of the head, the cartilage is eroded, calcified, and converted into endochondral bone (160-mm.), and then largely removed. Likewise, the periosteal shell on the tympanic aspect is rendered foraminous (183-mm.), then resorbed. When it has been rendered extensively perforate, the base becomes depressed (180-mm.), so that the roof of the cupola-like space is brought close to the floor. Over the floor of this marrow cavity endochondral bone spreads to produce an irregular, and later a fairly smooth layer (183-mm. and 180-mm., respectively). This change is the result of broadening and coalescing of originally separate islets of bone (183-mm.).*

The process of osteogenesis is complicated at this juncture by an act of developmental salvage, through the operation of which some bone of the "roof" is not removed, but, resisting erosion, remains to produce a peripheral rim, and in some specimens an intercrural crest on the obturator surface. The ridge may remain as the crista in the late fetus (210-mm., at cross) and in the adult (57-year, at cross). As is made clear by the formative stages (180-mm., 210-mm.), the relatively tall peripheral ledge of the base is composed of periosteal

*The discrepancy, in developmental advance, between the 183-mm. and 180-mm. stages is the result of the fact that the anatomical portions of each stapes do not develop at the same rate.

bone of the tympanic wall of the originally hollowed base and of endochondral tissue which makes up the inner lamella of the two-layered vestibular wall. When these strata meet and fuse, spaces of haversian character remain unobliterated, and persist to serve for transmission of blood vessels from the newly-formed submucosa into the substance of the base.

The longitudinal crista is similarly constituted; however, endochondral columns, joining the tympanic and the vestibular walls of the basal space, are more prominent than those around the circumference of the same cavity. In the course of this histologic remodelling of the base, the adjacent ends of the crura are altered in form. So variable is the extent of the change that at the points of crural implantation there may be great differences in spread. Usually the area of anterior attachment is the smaller of the two, corresponding to the reduced size of the anterior crus. When a crest is present on the base, it is more likely to be continuous with a free margin of each crus rather than to exist as a segregated ridge. Mucous membrane, pressing medialward to invest the outer wall of the otic capsule, covers the tympanic aspect of the stapedial base. Here, as in the case of either crus or head, it rests upon bone which is chiefly of endochondral derivation.

CONCLUSIONS AND SUMMARY

The very form of the cartilaginous model out of which the adult stapes is made sets the peculiar developmental problem which the ossicle must surmount. This form represents an early modification, in the human embryo, of the annular mass of cartilage, branchial in derivation, which constitutes the stapedial primordium; it is one which offers a flattened base to the vestibular window of the otic capsule, and a cylindrical, foveate head to the medial articular extremity of the incus. When ossification begins in the stapes (150-mm. fetus) it is initiated in a single center on the obturator (tympanic) surface of the base; spread occurs from that solitary center around the corresponding aspect of the crura and the head (160-mm.), so that periosteal bone is continuous circularly (183-mm.). Concurrently, the crura are gradually enveloped by bone; but bone extends on the base only as far as the lipped margin. At the same stage, too, bone clasps the medial margin of the cylindrical head, the main bulk of the capital end being as yet unaltered.

Beneath the periosteal shell of the whole ossicle cartilage is eroded, calcified, and converted into endochondral bone, the obturator surface being perforate for the transmission of vascular osteogenic

buds which produce these changes. The localization of these foramina is significant; they are concentrated on the obturator surface, and their widening and coalescence (183-mm.) result in removal of that entire aspect of the head and the crura (245-mm.), and to a considerable extent, of the corresponding surface of the base. In the basal portion of the stapes some of the periosteal bone is, as it were, salvaged; as if it were a tent-like structure undergoing collapse, the periosteal roof sinks to fuse with the endochondral floor (180-mm., 210-mm.). The osseous part of the base is, therefore, of double origin: the marginal elevation and the inconstant crista (245-mm., term) are remnants of originally separate lamellae. Comparably, endochondral bone of the head, abundant in the midterm stapes (180-mm.), is largely removed in later stages (245-mm., term) to leave a smooth capital interior; it occasionally persists in the form of a ledge (term; adult). In either case, the articular surface of the head is rendered bilaminar, since cartilage is lined by a lamella of endochondral bone. During the progress of these profound alterations in form and structure, the periosteum does not work perceptibly to increase the dimensions of the stapes; epiphyseal areas of growth are wanting.

Lacking the customary mechanism for expanding and lengthening, once bone formation begins in the midfetal period, the stapes ceases to increase in size; the ossicle has attained adult dimensions while the remainder of the body is still fetal. The transformation which converts the crura into channelled columns, the head into an excavated cylinder with a bilaminar articular extremity, and the base into a thin, two-layered plate is of the nature of a remodelling. This process results in striking reduction in bulk. The several parts are excavated, and the space thus exposed made continuous circumferentially to face the obturator foramen—the latter space being greatly enlarged at the expense of that which originally contained marrow and endochondral bone. The surface of bone thus exposed by erosion of the "diaphyseal" wall is draped by mucous membrane; blood vessels in a submucosal position communicate with others which course peripherally in the endosteal bone of the head and of the base.

The base of the stapes is closely accommodated to the vestibular window in the capsular wall, the dimensions of the fenestra being fixed during the fifth fetal month. The head articulates with the incus; the latter bone, like the malleus and unlike the stapes, adheres to a conventional pattern of bone development. Of the three bones in an ossicular chain only the stapes is the anatomic nonconformist.

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V

RECURRENT SWELLING OF THE PAROTID AND SUBMAXILLARY GLANDS FOLLOWING BRONCHOSCOPY

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Interest in the case presented in the following discussion was aroused because of an abnormal swelling of the salivary glands associated with bronchoscopic aspiration. Investigation of the patient's history revealed that following every bronchoscopy for over a period of four years this unusual condition had recurred consistently.

Since no reference to this type of abnormal swelling could be found in the literature, the writer has attempted through experimentation to find a logical explanation. Consequently this discussion will consist of the presentation of one case illustrating the swelling of the salivary glands following bronchoscopy, and a brief review of the applied neuro-anatomy, the physiology, and the pharmacological action of the drugs used in the investigation. An explanation of the phenomenon is tentatively suggested.

REPORT OF A CASE

N. K., a white male, aged 68 years, weighing 185 pounds, had been bronchoscoped at intervals of seven days for a period of four years. From the history, the patient had had a chronic productive cough and bronchial asthma for four years. Improvement had been very gradual. The writer first saw the patient in July 1941 at a time when the asthmatic attacks were very mild and infrequent. However, the patient still complained of a moderately productive cough and of a rather distressing swelling at the angles of both jaws and under the midportion of the lower jaw on each side of the neck for from two hours to two days following bronchoscopy.

Presented as a candidate's thesis to the Detroit Oto-Laryngological Society, December 1943.

From the Department of Laryngology, Rhinology and Otology of the City of Detroit Receiving Hospital and Wayne University School of Medicine, Detroit, Michigan.

Physical examination was essentially non-informing. On inspection one found only that both eyes had been needled for cataracts. The pupils were small and very irregular. Vision was only fair even with correction. The chest was clear to percussion and auscultation. The heart was not enlarged and there were no murmurs. The abdomen was soft and no tumor masses could be felt. The extremities were negative. The reflexes were physiological. The blood pressure was 165/85; temperature, 98.6° F.; and respiration, 18. Palpation of the radial and dorsal pedis arteries revealed a moderate arteriosclerosis for one his age. The laboratory findings were normal.

The first bronchoscopic examination by the writer was done on July 24, 1941. The following findings were noted: a direct laryngoscopic examination revealed a moderate amount of congestion about the larynx and some thickening of the mucosa, but no ulceration; the vocal cords met in the midline and their edges were smooth. There was very little secretion in the trachea. A 7-mm. bronchoscope was passed and the following observations were made. The mucosa of the trachea was pale pink in color. There were no protrusions, and no displacement of the trachea was noted. The carina was in the midline and the mucosal surface was pale. Small flaques of grayish-white, tenacious secretion adhered closely to the mucosa. The right stem bronchus at its orifice was definitely narrowed; the mucosa was pale and thrown into irregular folds, chiefly longitudinal, with a slightly nodular roughening of all surfaces. The orifices of the secondary divisions were definitely narrowed and contained secretion similar to that previously described, but in smaller amounts. Each orifice was dilated with an aspirating dilator. The left stem bronchus was very similar to the right except that there was some thickening in the lower part. The findings were definitely those of a tracheobronchitis associated with a tracheobronchial spasm.

The author was reluctant to accept a previous diagnosis of bronchiectasis without confirmatory bronchograms. These, however, were not made immediately since the writer wanted to study the case further in connection with the amount of secretion present. Additional bronchoscopies were done at intervals of seven days. Each time the patient was bronchoscoped there occurred consistently a marked swelling of the salivary glands and particularly of both parotid glands.

During the six weeks following the first bronchoscopy, there was a marked increase in the quantity of mucopurulent material aspirated; however, the patient stated that there was much less cough-

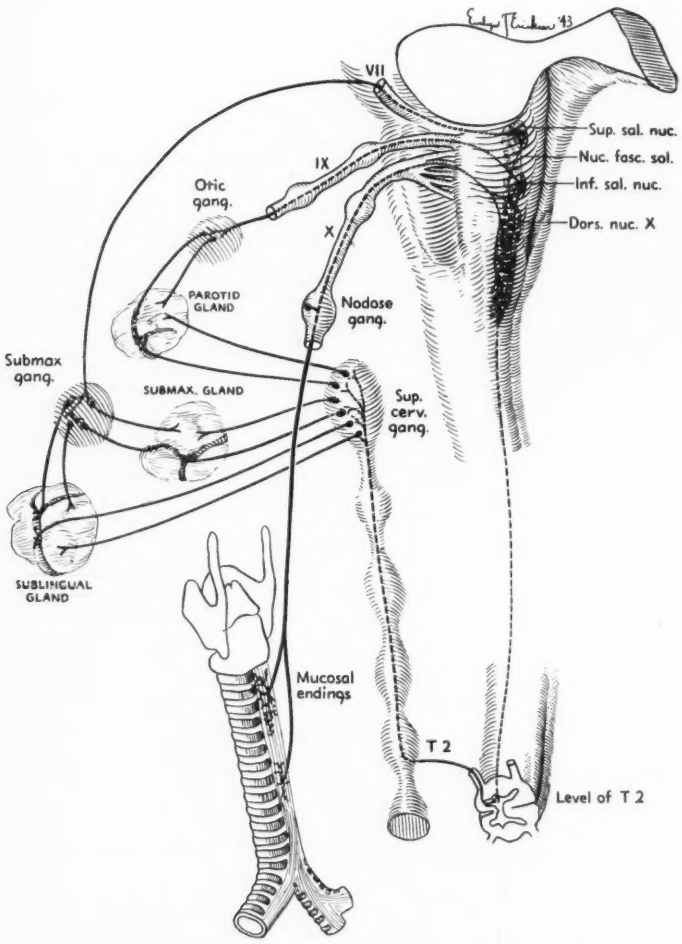

Fig. 1.—A representation (without scale) of the proposed path of the reflex as discussed in the text: A.—Stimulation of the mucosal endings in the sensory field of the trachea produces an afferent impulse via the ganglion nodosum of the vagus to the nucleus of fasciculus solitarius, across a synapse, to the salivatory nuclei. B.—The motor response may be either: (1) via the superior salivatory nucleus, thence to the facial nerve, to the chorda tympani, to the submaxillary ganglion, across a synapse, and finally to the submaxillary and sublingual glands; or (2) via the inferior salivatory nucleus to the glossopharyngeal nerve via the tympanic ramus to the otic ganglion, across a synapse, and finally via the auriculotemporal nerve to the parotid gland.

ing than at any time for the past four years. Two months after the writer's first examination bronchograms were done. These showed a small but very definite bronchiectatic area in the middle lobe bronchus of the right side. Subsequent bronchoscopic studies revealed a marked general improvement. While the improvement was entirely satisfactory, every bronchoscopic aspiration was accompanied by this unusual swelling of the salivary glands.

ANATOMY

The innervation of the salivary glands is well known. The autonomic nervous system is an efferent system and consists of two systems that are antagonistic or complementary. They are referred to as the sympathetic and parasympathetic nervous systems. Kuntz[1] has traced the conduction pathways of the autonomic nervous system in connection with the innervation of the salivary glands and the tracheobronchial tree. He describes briefly preganglionic and ganglionic neurones of both the sympathetic and parasympathetic nervous systems as follows:

"Autonomic Innervation of the Salivary Glands

"Sympathetic—Preganglionic neurones: Cells in the intermediolateral column in the spinal cord send fibres via the upper thoracic white rami and sympathetic trunk to the superior cervical ganglion. Ganglionic neurones: Cells in the superior cervical ganglion send fibres via the plexuses on the external carotid and external maxillary arteries to the submaxillary and sublingual glands and via the plexus on the internal carotid artery to the parotid gland.

"Function: Augmentation of secretion.

"Parasympathetic—Preganglionic neurones: Cells in the superior salivatory nucleus send fibres via the facial nerve, chorda tympani, and lingual nerve to the submaxillary ganglion. Cells in the inferior salivatory nucleus send fibres via the glossopharyngeal nerve and its tympanic ramus to the otic ganglion. Ganglionic neurones: Cells in the submaxillary ganglion send fibres directly to the submaxillary gland and via the lingual nerve to the sublingual gland. Cells in the otic ganglion send fibres via the auriculotemporal nerve to the parotid gland.

"Function: Augmentation of secretion." (Fig. 1.)

Sensory supply to respiratory tract—Afferent fibres from the lungs and the bronchi via the vagus with cell bodies in the ganglion nodosum send fibres to the tractus solitarius and the surrounding area of the lower medulla.

Function: Sensation from the trachea and the bronchi. (See Fig. 1.)

PHYSIOLOGY

When one takes into consideration that many things have been known to alter the normal secretory action of the salivary glands, one is unquestionably impressed with the complexity of salivary secretion. Mullin[2] has described the cooperative action of blood vessels, epithelial glands, afferent and efferent fibers of various cranial nerves in harmony with a central mechanism by which these are excited to coordinated action.

Physiologists and pharmacologists have shown by animal experimentation that by active stimulation of any sensory (afferent) nerve there may be an increase in salivary action. The act of mastication causes an increase in the flow of saliva but the actual rate of secretion is directly proportional to the type of food. One would expect, therefore, that hydrogen ion concentration tests would vary widely. Mullin[2] stated that Weber has shown "that after the application of different stimuli the reaction of the saliva varies, rapidly changing from a weak acid to neutral, but only occasionally becoming alkaline."

Not only is it possible to excite the salivary glands to increased activity by artifically stimulating afferent nerve fibers supplying them, but it is also possible to produce the phenomenon by stimulation of the central end of the vagus. Not infrequently stimuli arising from pathological conditions in the esophagus may be responsible for a profuse salivation. The writer has seen one such case in which this phenomenon was produced by esophagoscopy and was associated with a swelling of the salivary glands similar to the case described in this paper.

PHARMACOLOGY

The principal drugs used in these experiments were atropine, mecholyl, and epinephrine since they produce the desired effects upon the parasympathetic and sympathetic nervous systems. A brief review of their action is included.

According to Sollman[3] atropine produces various peripheral and central effects. These may be summarized as follows:

(a). Atropine produces a complete paralysis of the myoneural junction of the parasympathetic nerves—viz., secretory, bronchial musculature, cardiac and intestinal vagus, oculomotor, and uterine.

(b). Atropine produces an action antagonistic to that of pilocarpine, physostigmine, and muscarine.

(c). Atropine in small doses produces a slight stimulation while in larger doses it produces a paralysis of smooth and cardiac muscles and other cells.

Sollman also stated that the dosage required to paralyze the parasympathetic response varies widely with the different organs. He has shown this experimentally and reports his findings in terms of mg. per Kg. The dosage required to block the response to an electrical stimulation was found to be as follows:

"Salivary secretion (Chorda), 0.025 to 0.15

Cardiac vagus 0.07 to 0.7

Pyloric sphincter, small intestine, chorda dilator to 5 mg.

Urinary bladder more than 20 mg. per Kg."

He found, however, that very much smaller doses would prevent stimulation by pilocarpine.

The author has chosen a pilocarpine-like acting drug for the purpose of producing swelling of the salivary glands. According to Goodman and Gilman[4] mecholyl (acetyl-beta-methylcholine) has pharmacological properties that qualitatively are similar to those of acetyl-choline except that the former compound is more stable and lacks nicotinic effects on autonomic ganglia. They have demonstrated experimentally that mecholyl produces increased sweating, salivation, lacrimation, bronchoconstriction, as well as increased secretion of glands of the respiratory mucosa, and that both atropine and epinephrine overcome the bronchospasm. All glands innervated by cholinergic nerves are therefore stimulated. Goodman and Gilman also classify nerves as to whether or not they release acetylcholine or an ephedrine-like substance at their terminals. Cholinergic nerves release acetyl-choline at their terminals; these include most postganglionic parasympathetic fibers, autonomic preganglionic fibers whether sympathetic or parasympathetic, splanchnic (preganglionic) fibers to the adrenal medulla, the sympathetic fibers to the sweat glands, and the somatic motor nerves to skeletal muscles. Adrenergic nerves would include all nerves whose impulses act through the release of an ephedrine-like substance (adrenine, sympathin) and consist only of postganglionic sympathetic fibers.

Sollman[3] has pointed out that the typical action of epinephrine consists in a "highly specific stimulation of the receptive mechanism

of the entire sympathetic system." The inference then is that the effects upon any specific organ would correspond entirely with the effects of stimulation of the sympathetic innervation of the organ in question. This is true with the use of ephedrine in ordinary concentrations; however, in very dilute concentrations the opposite effect may be noted. The most dramatic effect of epinephrine is, of course, the sharp rise in blood pressure. This is due primarily to a peripheral stimulation of the vasoconstrictor mechanism and of the accelerator mechanism of the heart. There is at first a slight drop in pressure which has been interpreted as being due to a beginning vasoconstriction. This is followed by a slight secondary rise which has been ascribed to a stimulation of the vasoconstrictor center superimposed on the remaining peripheral vasoconstriction. The secondary and final fall in blood pressure has been attributed to impurities, presumably choline.

Sollman,[3] in his discussion of the effect of epinephrine on the heart rate of intact animals, stated that the drug seems to have a twofold action, namely: (1) acceleration because of direct cardiac stimulation; (2) slowing because of reflex vagus stimulation. The inference, therefore, is that when the vagi are intact and in good tone, epinephrine in ordinary doses would slow the heart rate and that when the vagal tone is low a quickened heart rate would result. The vagus effect is purely central since it is well known that the cardiac rate is not slowed by cutting the vagi and that perfusion of epinephrine through the medullary centers also slows the heart rate.

Best and Taylor[5] have pointed out that epinephrine provokes secretion of the salivary and lacrimal glands. These authors have also shown that increased secretion of the submaxillary glands is accompanied by increased blood flow. Sollman[3] stated specifically that nerve trunks are not affected by epinephrine.

EXPERIMENTATION

In an attempt to discover the nature of this unusual swelling of the salivary glands, drugs were used first to stimulate and then to annul the autonomic effects. This seemed the logical method of approach because to date there is no satisfactory generalized sympathetic depressant. Therefore, the most satisfactory method of studying the role of the sympathetic nervous system was to use a drug to annul the action of the parasympathetic. Atropine does this well.

EXPERIMENT 1. On each of four occasions the patient was given atropine sulphate hypodermatically about 45 minutes before the

routine bronchoscopic aspiration. The doses used were: 1/150 gr.; 1/100 gr.; 1/75 gr.; 1/50 gr. A typical atropine reaction was noted on each occasion. The drug used in the stated doses did not prevent the swelling of the salivary glands. Because of a slight increase in the intra-ocular tension (OS tension 40; OD tension 26) atropine in a dose greater than 1/50 gr. was not used.

The experiment shows that the doses of atropine used within limits of the patient's tolerance did not block the reflex responsible for the swelling of the glands. Because of the increased intra-ocular tension only inadequate doses of atropine were possible. Consequently, it seemed logical that the opposite of parasympathetic inhibition would make a more appropriate check. The inference, therefore, is that if by the use of a powerful parasympathetic stimulant this characteristic swelling of the salivary glands occurs, then one has produced the same reflex by the use of certain drugs that is produced mechanically by the insertion of the bronchoscope into the tracheobronchial tree.

EXPERIMENT 2. As previously stated mecholyl is a powerful parasympathetic stimulant and profuse salivation is observed shortly after its administration in ordinary doses. In this experiment, 1 cc. contained 0.025 gm. of mecholyl. The patient was given 0.6 cc. or 0.015 gm. of this preparation subcutaneously. This dosage produced an excessive flow of saliva in a very few moments accompanied by a definite, prominent swelling of both the parotid and the submaxillary glands. The swelling was synchronous with the salivation and lasted for a period of two hours. The inference, therefore, is that the same reflex has been produced by stimulation of the parasympathetic nervous system as was produced by mechanical stimulation of the tracheobronchial tree with the bronchoscope.

EXPERIMENT 3. In order to evaluate the role of the sympathetic nervous system it remained only to stimulate the system with a powerful agent such as epinephrine. If, after using such a potent sympathetic stimulant as this, one finds that there is an increase in the flow of saliva unaccompanied by swelling of the salivary glands one can feel with reasonable certainty that the unusual swelling of the parotid and submaxillary glands must be due to a stimulation of the parasympathetic nervous system.

The writer gave the patient 6 m. of epinephrine (1:1000) hypodermatically. The blood pressure, the pulse and the respiration were noted just prior to the administration. They were as follows:

blood pressure 165/85; pulse 88; and respiration 18. Following the administration of the drug there was a sharp increase in the blood pressure. The highest point reached was 190/40. A gradual increase in pulse rate to 110 per minute was observed, but the rate gradually returned to normal in 15 minutes. The respirations became deeper, slightly irregular, and only moderately increased in rate to 24 per minute. These observations together with a general flushing of the skin, an obvious fullness of the peripheral vessels, slight salivation and lacrimation gave a typical reaction to epinephrine. Outstanding is the fact that even though there was an increase in the flow of saliva there was no swelling of the salivary glands. Subsequent bronchoscopy produced the usual typical swelling.

EXPERIMENT 4. In order to evaluate properly all the facts at hand one must consider the role of anesthesia, both local and general. Cocaine 10 per cent was used for topical application to the pyriform sinuses just before each bronchoscopic aspiration. On several occasions, however, this procedure was omitted and the characteristic swelling of the salivary glands was noted. The inference is that there was no specific reaction to cocaine involved in the response of the salivary glands to bronchoscopy.

The writer did not give the patient any general anesthetic. However, this same swelling of the salivary glands was noted in another case in which esophagoscopy was performed after the patient was given pentothal sodium intravenously. The inference, therefore, is that the reflex is not abolished at a safe level of surgical anesthesia.

The writer would not expect the salivary response to be lost under anesthesia since the salivary nuclei lie at the same anatomical level as the cardiovascular and respiratory centers. The latter are not abolished by anesthesia. Psychic mechanisms may be ruled out by the persistence of this response during anesthesia.

EXPERIMENT 5. For the explanation presented in this discussion it is necessary to eliminate the possibility of the effect that position and mastication might have on the production of the swelling of the salivary glands. Salivation and swelling of the glands were tested by having the patient lie down with his head in various positions and by having the patient eat various types of food. Neither of these factors produced the swelling. Mastication did cause an increase in the flow of saliva and the rate of flow appeared proportional to the taste of the food. These responses can be considered only as normal physiological ones.

TABLE 1.—SUMMARY OF EXPERIMENTAL FINDINGS.

DRUG	ACTION OF DRUG	RESPONSE OF SALIVARY GLANDS OF PATIENT	SALIVARY GLAND RESPONSE TO BRONCHOSCOPY
Atropine	Parasympathetic Paralysis	No Change	Swelling
Mecholyl	Parasympathetic Stimulation	Increased Salivation and Swelling	No Further Swelling
No Satisfactory Generalized (Ortho) Sympathetic Depressant			
Adrenalin	(Ortho) Sympathetic Stimulation	Increased Salivation No Swelling	Swelling
Cocaine to Pyriform Sinuses and Trachea	Anesthetic Agent to Sensory Field	None	Swelling
General Anesthesia: Pentothal Sodium	Psychic Depressant	None	Swelling

Since neither posture nor the act of mastication could be shown to produce the swelling, the inference is that the passage of the bronchoscope into the tracheobronchial tree is the only mechanical factor that could have produced this unusual swelling of the glands.

SUMMARY. The action of the drugs used, the salivary gland response to these drugs and to bronchoscopic aspiration is shown in Table 1.

DISCUSSION

In Experiment 2 the author has demonstrated that the same swelling of the salivary glands could be produced by stimulating the parasympathetic nervous system with mecholyl. The writer has shown to his own satisfaction that stimulation of the sympathetic nervous system with such a powerful agent as epinephrine neither produced the swelling nor prevented it during immediate subsequent bronchoscopic aspiration. The nature of the response therefore, in all probability, is via the parasympathetic nervous system.

The author does not know the relative value of local vascular versus cell changes in the glands and therefore will assume that both vascular and cell changes play a part until proved otherwise.

The writer questions the anesthetic value of cocaine as a complete block to the reflex below the level of the larynx since cocaine at this level acts only as a depressant and not as a complete local anesthetic. Because of the narrow margin of safety afforded by cocaine, complete anesthesia of the entire tracheobronchial tree is impossible.

It was demonstrated that neither mastication nor the position of the head was responsible for the swelling. Obviously then, there were no mechanical factors other than the passage of the bronchoscope to be considered in these experiments. Sialograms, read both by the writer and by Dr. J. C. Kenning, chief roentgenologist at Receiving Hospital, did not reveal the presence of either intrinsic or extrinsic mechanical obstruction to the ducts.

The proposed path of the reflex is illustrated in Fig. 1.

CONCLUSION

A case is presented of abnormal swelling of the salivary glands in response to mechanical stimulation of the trachea. The effects of various autonomic drugs have led to the tentative conclusion that it is a parasympathetic reflex mediated by a sensory path over the vagus nerve and a motor path over the seventh and ninth nerves. The reflex centers involved are in the medulla.

The writer is indebted to Miss Evelyn T. Erickson for the illustrative drawing.

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VI

OUR CHANGING CONCEPTION OF ACUTE LARYNGOTRACHEOBRONCHITIS

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Since the pandemic of influenza in 1918, many cases of acute laryngotracheobronchitis have been reported under various terms. At first the picture was not clear as the condition followed both measles and influenza. It seemed to represent a phase of upper respiratory infections which vary so tremendously from year to year. Until recently the term "fulminating" was the most descriptive, as a good result could only be looked upon as an accident rather than the result of good care. Fortunately, that is no longer true.

SURGICAL PERIOD

The period between 1918 and 1938 might quite logically be called the surgical or laryngological period. Because of the overwhelming seriousness of the obstructive symptoms, the disease was looked on as belonging primarily to the realm of the laryngologist. Besides relief from the initial obstruction, it meant constant attention on the part of the laryngologist to relieve recurring obstruction lower down in the respiratory tract. Reports centered around the question of whether intubation or tracheotomy was preferable and whether the use of medication locally in the trachea and bronchi was an aid to the prevention of obstructing crusts. Supportive treatment was most essential and was emphasized but it was always given a very secondary place. Notable contributions for this period were made by Baum,[1-3] Smith,[16] Gittens,[7] Jackson,[8] Richards,[13, 14] Neffson[9-11] and many others.

From the pathological point of view, these cases are fundamentally the same as the streptococcus infections in the influenzal cases of 1918. When staphylococcus aureus is the organism recovered, the necrosis to be noted in the larynx and the trachea is apt to be more pronounced, while it is more superficial when the influenza

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bacillus is the recovered organism. In view of the pathology it seems unwise to refer to any of these cases, even the milder ones, as laryngitis alone. The only patients in this series which fall into this group died from profound inflammation of the glottis, presumably before it had time to extend to the trachea and the bronchi.

The following three cases, briefly cited, will illustrate the conception which most of us had of this disease.

CASE 1.—This case occurred in a little girl of four in the winter of 1931. She was seen by a pediatrician because of a cold. Two days later, while in the neighborhood, he stopped in to see her as a convenience to himself. While he was there, she developed marked difficulty in breathing. He immediately put her in his car and rushed her to the hospital. On the way to the hospital it was necessary to stop the car and start artificial respiration. On admission to the hospital she was a very sick child with profound retraction and so-called pale cyanosis. The air exchange of the lungs was very poor. A tracheotomy was done with immediate relief. The patient was put in the steam room and given the usual infusions of glucose and saline, and transfusions of blood. Three days after the tracheotomy, the child became restless and respirations increased. No breath sounds were getting through on the left side of the chest. A bronchoscope was passed and the left bronchus was seen to be plugged with exudate which could not be removed by the suction tube. This exudate appeared like pieces of felt and had to be removed by forceps. During the next 48 hours it was necessary to do six more bronchoscopic treatments for the removal of plugs in the main bronchi, first on one side and then on the other. After the removal of these plugs there was no purulent exudate in the smaller bronchi. Cultures of the throat on admission showed the influenza bacillus in pure culture.

CASE 2.—This case occurred in a boy of 11 months in the winter of 1932. The boy had been under the care of a pediatrician and two weeks before admission had been given 10,000 units of diphtheria antitoxin as part of a health drive in his town. For three days the boy had had a cold with cough and croupy breathing but nothing alarming. He awoke from a nap fighting for breath and was rushed to the hospital immediately. There was very marked retraction, no cyanosis, but the child appeared very toxic. Aeration on both sides of the chest was poor. A laryngoscopic examination showed very marked redness and edema of the laryngeal mucosa so a tracheotomy was done. The patient did not get marked relief. Twelve hours

after being in a steam room the patient developed such marked dyspnea that it was necessary to do a bronchoscopic examination. Crusty tenacious secretions were removed from both main bronchi. Four hours later is was necessary to do another bronchoscopy. Eight hours after this second bronchoscopic examination there was an attack of dyspnea accompanied by definite cyanosis. This time, after removal of the crusts from the main bronchi, it was seen that there was purulent exudate in the smaller bronchi and this could only be removed by the suction tube as it was impossible to pass the bronchoscope deeper. Two more bronchoscopies were done during the next twelve hours but neither gave the child relief in breathing. Examination of the chest showed the typical picture of bronchiolitis. Clinical cultures showed the influenza bacillus and pneumococcus, type III. Postmortem cultures showed only staphylococcus aureus in combination with nonhemolytic streptococcus. Edema was not marked in this case despite the age of the patient. The outstanding feature was the rather extensive involvement of the bronchioles and the lung parenchyema—much the same picture as that seen postmortem in adults. There was no evidence of pneumothorax or mediastinal emphysema. This is noted as the question has been raised whether this complication did not account for the poor results in these patients rather than an obstruction in the lower air passages.

Case 3.—This case occurred in a little girl aged four in the winter of 1933. A pediatrician was called at 3:30 P. M. as the child complained of a sore throat. The child vomited about 5:00 P. M. and felt better. At 6:30 P. M. the pediatrician was called a second time as he had not yet arrived to see the patient. At 7:00 P. M. he was called again, not because the child seemed terribly sick, but because the parents were getting annoyed as he had not shown up. At 7:10 P. M. he was called and the parents said the child was dead. It seems the patient had had another attack of vomiting, had choked and become black. The heart was not beating when the pediatrician arrived and a tracheotomy was unsuccessful. Autopsy showed acute laryngitis with tremendous edema of the epiglottis and the larynx. There was pulmonary emphysema and edema of the lungs but no definite inflammation of the lower trachea and bronchi. Cultures made from the heart blood and from the lungs showed a pure culture of H. influenzae, type B.

CHEMOTHERAPY PERIOD

Beginning with the extensive use of sulfonamides, the picture of acute laryngotracheobronchitis has changed. It may change again

with the advent of penicillin. As the earlier period was designated the laryngological period, the present period beginning about 1938 might be designated the pediatric period. Supportive or medical treatment which previously was given a very secondary position has now assumed a more prominent role. We have not had a severe influenzal year for some time but we do see enough severe cases to realize the change in the picture. Whether these observations will hold up in the face of a severe influenza epidemic cannot be stated.

In 1941, Sinclair[15] from the Department of Pediatrics at New Haven, reported ten cases of acute laryngotracheobronchitis with septicemia due to the Haemophilus influenzae, type B. Eight of these occurred between January 1940 and April 1941. The other two occurred during the preceding seven years, which is not a fair comparison since blood cultures were not done routinely then. If we use tracheotomy as the indication of a very seriously sick patient—and it may not be entirely correct to do that—there were two out of the eight patients who did not require a tracheotomy.

Sinclair noted that the appearance of shock seemed to be out of all proportion to the short duration of the obstructive symptoms. After a tracheotomy was done, most of these failed to show immediate improvement. As the respiratory distress was gradually relieved the patients appeared prostrated and the temperature remained elevated. It has been the distinct impression of the pediatricians that these cases constituted the most seriously sick group. The other observation which Sinclair made on his ten cases was that those receiving adequate sulfonamide therapy recovered and those not receiving it (four in number) died. The use of anti-haemophilus influenzae type B rabbit serum was used on one patient who could not tolerate the sulfonamide and since then it has been used routinely in addition to sulfadiazine.

Immediately upon admission the patient is examined. If it is not necessary to do an immediate tracheotomy he is placed in the steam room (humidity 80%, temperature approximately 80° F.) and sulfadiazine is started. If possible this is given by mouth; otherwise, the sodium salt is given in normal saline, to be followed by an infusion of saline and glucose. Dosage is figured at the rate of one grain per pound per day, with half of this amount being given as the initial dose. This dosage is usually enough to keep up the blood level to the neighborhood of 10 to 12 mg. per 100 cc. Blood transfusions are occasionally given but not to the extent previously used. When a tracheotomy is necessary, it is usually necessary on admission or very shortly thereafter. Since the indications for tracheotomy depend

upon clinical experience, they will vary in every clinic. Here they have been done in slightly over 20 per cent of the cases, which is higher than Neffson's figures but much lower than those of some clinics.

Since the treatment of these patients is essentially the same as that of the 1933 period in all respects except for the use of the sulfonamides, any noticeable differences should be considered as resulting from the use of these drugs. Possibly another severe influenzal year will change the picture somewhat but that seems unlikely.

At the time of the summation of these results, December 1, 1943, the epidemic of mild influenza had not appeared. That epidemic had not produced a noticeable increase in these cases by January 1944. In the winter of 1941-1942 there were 24 cases of laryngotracheobronchitis with two deaths, both from pneumothorax and mediastinal emphysema, within 12 hours after admission. In the winter of 1942-1943 there were 30 cases with one death. This patient had a hemoptysis and died during a bronchoscopic examination within one hour of admission. Beginning the winter of 1943 there were 19 cases up to December 1—more than twice as many for the same period of the two previous years. There was one death—the very last case—which only served to spoil perfect statistics. This patient had mediastinal emphysema but did not die until the sixth day. Then the statement made by Sinclair in 1941 can be confirmed, since everyone of these 73 patients who received adequate sulfonamide therapy recovered, with the possible exception of the last patient. This is a very striking feature in comparison with the mortality rates of 50 per cent and over for the earlier period though it has not been confirmed in many other clinics. Neffson reported nine deaths in 42 patients receiving adequate sulfonamide therapy but does not state whether any operative accidents were included in this group.

During the two winters, 1941-1943, there were six cases of tension pneumothorax and mediastinal emphysema. Five of these patients had had a preliminary bronchoscopy or the bronchoscope was passed in order to facilitate the tracheotomy. Instead of eliminating respiratory effort during a tracheotomy and hence preventing this accident from happening, these figures would suggest that it is the cause of the trouble. Many laryngologists deny this and they may be correct since there were two additional cases this fall following a tracheotomy by the standard technic. Suffice it to say that in the great majority of cases the less instrumentation, the better and the usual technic for doing a tracheotomy is sufficient. These cases, if we are to evaluate any series accurately, should be regarded frankly

for what they are, namely, operative accidents. No one is to be blamed for an operative accident since these are all such terribly acute emergencies.

There is one other very striking feature resulting presumably from the extensive use of the sulfonamides. The constant recurrence of obstruction lower down in the respiratory tract in the tracheotomized patients and illustrated in the first two cases cited was an outstanding feature in the earlier cases. In 16 of this series of 73 cases, a tracheotomy was necessary. In none of these cases did obstructing crusts develop, requiring even a single bronchoscopy, except in the last case. This refers to those obstructing crusts which can not be removed by a suction catheter passed through the tracheotomy tube. It is not to be expected that this will always hold true, as the very last case spoiled the perfect record but there was the accompanying mediastinal emphysema to account for it in this one case. It has been suggested that these cases requiring repeated bronchoscopies after the tracheotomy tend to occur in cycles so only a severe influenza epidemic will tell whether this apparent result of the sulfonamides is universal.

There is one other phase of this disease which it seems worth while to discuss, namely, etiology. Here in New Haven blood cultures are made on admission of the patient along with cultures from the nose and throat. Besides the usual blood agar plates for culture, the swab from the nose is placed in rapid typing broth. This swab is used as it is felt to have less contaminants than that from the throat. Within 12 hours the pathogens have outgrown the nonpathogenic organisms. They can then be typed against specific sera. Within 12 hours it is possible to tell with what pathogenic organism one is dealing and that is a big help in handling these patients. Of course, cultures are made for the Klebs-Loeffler bacillus, too. Excluding the latter, the pathogens which are commonly found in the upper respiratory tract are the hemolytic streptococci, the haemophilus influenzae, the pneumococci which used to be lumped together as type IV, and the staphylococcus aureus. The last organism is very commonly found on the skin while the others are confined to the mucous membranes.

Referring once again to Sinclair's article, you will recall he reported eight cases of H. influenzae type B septicemia in the laryngotracheobronchitis series for the winter of 1940-1941. Many strains of the influenzae bacillus occur in the upper respiratory tract. In 1931 Pittman[18] demonstrated that the pathogenic strains are usually encapsulated and that the capsules have a specific soluble antigen

which renders them capable of differentiation into the serological types (A to F). The majority of strains isolated from "influenzal meningitis" are type B. Sinclair showed that the cases of septicemia with laryngitis also belong to this type.

Both Neffson[9-11] and Davies[4] emphasized the frequent occurrence of the staphylococcus aureus. However, Davies pointed out that this was true in the fatal cases, especially in those patients under two years of age. It is true in patients having tracheotomies and bronchoscopies. It can readily be understood how this organism is introduced during frequent suctionings. It is a frequent cause of death, but occurring so late and obviously as a contaminant, it can be dismissed as a factor in the natural history of the disease.

In going over our own series since Sinclair's report we find the following. In the winter of 1941-1942 there were four cases of septicemia due to H. influenzae type B and during the winter of 1942-1943 there were no cases. Instead there were a great many pneumococci, type VI and above. For the most part these are found in rather mild cases. This fall there were twice as many cases as we had seen in the corresponding period last fall but it was not till the fifteenth case that an H. influenzae type B septicemia was found. The number of infections with staphylococcus aureus and hemolytic streptococcus have been insignificant and have occurred only in conjunction with other pathogens, usually the pneumococcus. There have been no septicemias except those caused by the H. influenzae type B. The last case, referred to as the one which only served to spoil a perfect series, probably belonged to this series of H. influenzae type B septicemia group. Cultures made the day after admission were all negative, yet cultures of the nose and throat made two days later showed pure cultures of H. influenzae type B. Postmortem cultures apparently are a war casualty. There was still a third case of H. influenzae type B septicemia requiring a tracheotomy which occurred before this report was made but was not included in this series. These cases would indicate the probability of many more this winter.

Included in our series are a certain number of patients who have relatively little fever and a low white blood cell count and in whom no pathogens are found on culture. After the initial respiratory difficulty subsides, the sulfonamide is stopped and they are well within three or four days. Does this represent a virus infection without secondary invaders? No one seems able to say definitely how much clinical evidence of inflammation is produced by the virus alone. It seems worth while to point out that, while the virus may initiate the

process, the symptoms produced by the bacterial or secondary infection may not necessarily occur concomitantly. They may not appear for one or two weeks. This directs most of the attention to the bacteria as the etiological agent which it is necessary to combat in this disease.

When pathogens are present the case is apt to be more severe, yet this may not be striking especially where the pneumococci are concerned. The staphylococcus aureus is not commonly found early in the disease though it is frequently found late in the disease (especially in cases which have had instrumentation) and is responsible for a large number of deaths. As a true etiological factor it would seem to be excluded by these features.

In this series the hemolytic streptococcus was not an especially common pathogen, though this does not fit in with what was observed in the cases from 1918 on for several years. The one organism in this series which was found very early in the disease and which produced the most seriously sick patients was H. influenzae type B. This is an organism with such invasive qualities that it frequently invades the blood stream and causes the most extreme edema in the region of the glottis. I am going to suggest that this organism may bear a much closer etiological relationship to this disease than any of the other pathogens commonly found.

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VII

THE SIGNIFICANCE OF EOSINOPHILIA IN RHINOLOGY

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The nasal allergy problem is frequently a "bugbear" to the rhinologist as well as the allergist. Much investigative work has been done in the past three decades, but it must be admitted that some of the factors concerned have not been fully explained, either by the allergist or the rhinologist. Nor do I, at my kindergarten period in the field of rhinology mean to infer that I have already found the answers to these complex problems in some remote corner.

However, the finding of eosinophilia either locally, as in nasal smears, or in the blood stream, in the presence of one or many symptoms known to us all, has usually been associated with allergy. Before evaluating the probable significance of the finding of eosinophiles in the nasal chambers, we should review the normal location of eosinophiles and some of their variations in the body.[1, 2] The normal adult blood contains from two per cent to four per cent eosinophiles. The blood of children may contain as much as five per cent to six per cent eosinophiles normally. The number of eosinophiles is often higher in the morning than in the evening. Starvation may produce a fall in blood eosinophiles; also following various operative procedures there is frequently a fall in the blood eosinophiles. The eosinophiles are normally found throughout the gastro-intestinal tract, especially in the stroma of the mucosa; they are constituent elements of the bone marrow, thymus, lymph glands, and spleen, and occasionally are even found in the lung. A slight blood eosinophilia is frequently noted during menstruation.

In pathological conditions, eosinophilia has been found in patients with almost all of the known major types of inflammation and malignancies. Both local and general eosinophilia has been reported in occasional cases of tumors of the colon, carcinoma of the cervix of the uterus, sarcoma, carcinoma of the neck, and a great local eosinophilia has been reported in several cases of carcinoma of the stomach.

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Its finding in parasitic disease is well known. In trichiniasis, where it is very frequently found, cases have been reported without any increase in the blood eosinophiles. Local eosinophilia has been found in the bronchioles of asthmatics, in the colon in intestinal catarrh and in the lymph nodes in Hodgkin's disease. Both local and general eosinophilia has been found to follow injections of foreign proteins. Scarlet fever is the only exanthema that may be accompanied by marked blood eosinophilia; it may also occur after inoculation for scarlet fever. Other miscellaneous conditions reported associated with eosinophilia are chronic osteomyelitis, Paget's disease, pemphigus, psoriasis, bone tumors, malaria, leprosy, Addison's disease, gout, chronic nephritis, chronic colitis, duodenal ulcer, periarteritis nodosa, and rheumatic fever. A high pleural eosinophilia has been found in one per cent to five per cent of pleural effusions. It is even produced by drugs such as pilocarpine, camphor, salicylates, potassium iodide, and digitalis. It has been noted also that skin lesions which act destructively on the epithelium are frequently associated with a pronounced eosinophilia.

Familial eosinophilia is found often, although the family incidence of allergy and of transmissible parasitic disease doubtless accounts for some of the eosinophilias classified as familial. Thus, the list of conditions in which eosinophilia is found, either locally or generally, is so staggering as to suggest no possible single significance as to the real function or functions of the eosinophile. However, there must be some rationale to such a cell.

Colmes,[3] in his study of the comparison of the daily blood eosinophile count and the daily nasal eosinophile count in patients with hay fever, both before and during the season, discovered what appeared at first to be a somewhat confusing analogy. The eosinophile content of nasal secretions in no way compared individually with the blood eosinophile count, nor did either the local or the blood eosinophile count rise or fall with the severity of the allergic symptoms of each respective patient. However, as the hay fever season progressed, the group of patients as a whole did show an increase in nasal eosinophiles. Also as the pollen count of ragweed changed, by increasing or decreasing during the season, the severity of the symptoms and the blood eosinophiles seemed to follow the same type curve.

Thus, we have group parallels, but in many instances patients had marked symptoms and yet no rise in the nasal or blood eosinophiles. So, individually some of the results were confusing, but taken as a group the results seem to parallel what we should expect.

In 1941, from a series of 100 cases taken at random in which polypectomies were done here at the Massachusetts Eye and Ear Infirmary, polyps were sectioned and studied histologically. In every single polyp there was a marked eosinophilia just below the epithelium; this was less marked throughout the remainder of the polyp. This has no doubt been studied many times previously by others who have found a similar local eosinophilia. In 1929 Hansel[4] stated that the occurrence of eosinophiles in nasal secretions, together with an eosinophilic infiltration of the nose and sinuses, was an indication of allergy. Cohen and Rudolph,[5] in 1931, showed that the absence of eosinophiles in nasal secretions does not rule out the presence of allergy. This also has been verified many times.

Part of my purpose in presenting this paper is to offer an idea based on studies of the eosinophiles in relation to histamine which might throw some light on the function of the eosinophile. Many functions of the cell have been described. The major function of the eosinophile has been described as that of phagocytosis. Some believe the eosinophilic granules are actually phagocytized hemoglobin. There is evidence for and against this belief. Eosinophiles are thought by some to be a cell that takes part in the healing of tissue following disease or injury. These cells frequently disappear during an infection and reappear during the healing stage. The explanation for this has been that the eosinophile is delicately concerned with protein metabolism and any change in the body's protein may evidence itself in an increase in the eosinophiles, locally or in the blood stream. Postfebrile eosinophilia has been explained as the result of bone marrow stimulation by protein set free during the process of inflammation. As long as the inflammation persists the eosinophilic-leucocyte production is depressed. When the acute toxic stage has passed the myeloid tissue reacts to the stimulus of the protein products and produces an increase in the eosinophiles, thus giving a favorable prognosis. According to Schlecht,[19] eosinophilia is the expression of the reaction of the body against the toxic products resulting from the decomposition of foreign as well as native proteins. According to Baar[20] in 1927, eosinophilia indicates nonspecific sensitization to autogenous or foreign proteins. He said it was not necessarily related to the phenomena of anaphylaxis per se.

Now clinically, it has been proved time and again that in most allergic cases the injection of a moderate sized dose of histamine in a patient with asthma, or hay fever, or urticaria, who is in a quiescent stage, will produce an attack of asthma, hay fever or urticaria. It has been postulated that when the allergen meets the antibody a re-

action takes place on the surface of the cell. As a result of this reaction, a state of trauma is produced. This trauma is believed to release histamine. Histamine is a substance which was first isolated in 1910 by Barger and Dale.[6] It was later described as being formed by the action of putrefactive bacteria on histidine, one of the so-called essential amino acids[7] found in proteins necessary for the body's structure. Histamine has been isolated from intestinal contents; it has been found in variable amounts in different organs of the body, such as the lungs, muscles, heart, spleen, and intestinal and gastric mucosa. It is rendered physiologically inert by an enzyme called histaminase[8] which is present in abundance in the lungs, kidney, intestinal mucosa, spleen muscles, adrenals and blood. This is the basis for the use of histaminase (torantil) in the treatment of allergic rhinitis.

When histidine is decarboxylated in the breakdown of proteins of the body, histamine is released and this substance thereupon produces remarkable constrictor effects on smooth muscle, as in the arterioles and the bronchioles, and also produces a dilator effect on the capillaries; this latter action causes stagnation of the blood in the areas of reaction, and thus edema.

Anoxia raises the level of histamine in the blood stream. In acute shock the blood histamine is not raised, but on the fifth or sixth day following severe burns, for example, in the period of secondary shock when there is protein breakdown and absorption, the blood histamine level rises.[10]

Perhaps the most important work in the past few decades on histamine is that done by Code[11-14] in 1937. In certain conditions it had been discovered that the normal blood histamine[15, 16] content was increased. The object of Code's experiments was to determine in a study of the blood which portion carried the histamine. He studied the histamine equivalent of the serum, platelets, white blood cells and red blood cells of horse, rabbit and human blood. He found that of all the histamine noted in the blood stream of the horse, 95 per cent was carried by the white blood cell fraction, as compared to the red blood cells and the platelets. In rabbit blood, most of the white cells of the myeloid series contain eosinophilic granules in the cytoplasm. Here, too, as much as 87 per cent of the histamine of the blood was found to be carried by the white blood cell fraction. In human blood, the histamine activity was found to be as high as 77 per cent in the white cell fraction.

Then Code attempted to break down the white cell fraction into its component parts, first testing for the amount in lymphocytes,

then in monocytes and then in the polymorphonuclear cells. The blood of six patients with lymphatic leukemia was tested for histamine and compared with the normal blood histamine of 103 university students. The leukemia patients showed no more histamine in the white blood cell fraction of the blood which contained 93 to 99 per cent lymphocytes than the normal white blood cell fraction of the students' blood. Thus, lymphocytes were probably ruled out as the carrier of histamine. In sterile pleural exudates produced by irritation with petrolatum in rabbits, the greatest percentage of cells were found to contain monocytes; up to 90 per cent in fact. Here again there was no noticeable increase in the histamine content in the monocytes.

When myeloid leukemic cases were studied there was a tremendous increase in the histamine of the white blood cell fraction. Thus, Code concluded, that he had probably come upon the real carrier of the histamine. He then tried to find out which cell of the granular series carried most of the histamine.

Cellular deposits consisting of 70 to 80 per cent neutrophiles obtained from sterile exudates induced in dogs did not produce much histamine when the white blood cell fraction was studied. Therefore, the neutrophiles, at least in dogs, did not seem to be a source of histamine activity. In one human case studied, a predominantly neutrophilic leukocytosis did not show an increase in the blood histamine equivalent. These results seem to indicate that the neutrophiles are not usually concerned with the transport of histamine.

In rabbits, the granules in the polymorphonuclear cells are replaced by eosinophilic-staining granules and are called pseudo-eosinophiles. It was noted by Code, that the histamine content in rabbit blood is 50 to 200 times that of dog blood. In a sterile-produced exudate in rabbits, the cell count proved to contain 29 per cent so-called pseudo-eosinophiles and 11 per cent real eosinophiles; and here, the histamine equivalent noted was found greatly increased. Then MacDonald,[17] in England, studied the blood of four patients with eosinophilia. In every instance, the histamine equivalent exceeded the upper normal value of 0.05 r/cc. This all seemed very significant. Therefore, the first positive results obtained were with cells showing eosinophilic granules. According to Code, the accumulated evidence allows the conclusion that the eosinophile is, at least in most cases, a source in the blood of histamine activity. Histamine carriage is then apparently a function of the eosinophile.

Having arrived at this probable conclusion as to a function of the eosinophile as a histamine carrier, we might go a step further.

Knowing that in allergic reactions when the antigen, ragweed for example, meets the antibody, or reagin, there is a reaction consisting of edema and release of histamine due to this local trauma. The bone marrow is probably stimulated, perhaps by the excess histamine produced; eosinophiles are called forth from the bone marrow, pass through the blood stream to the site of the reaction. Knowing that the eosinophile is a histamine carrier, it seems possible that they are brought to the site of the reaction to carry off as much histamine as possible, probably a body mechanism to lessen the severity of the reaction. Some histamine may remain fixed in the tissues of reaction and thus make it difficult for the eosinophiles to take it all away. Depending on the type of reaction, whether intermittent or constant, the eosinophiles may be found in lesser or greater numbers in the blood stream or in lesser or greater numbers at the site of reaction.

In nasal polyps, therefore, there may be a continual presence of histamine due to repeated reactions caused by the antigen meeting the reagin in the nose, and thus we also find eosinophiles in nasal polyps in more or less greater numbers. The further proof of this possibility lies in work that must still be done. The problem is: "Is there more histamine always present in nasal polyps as compared with other tissues of the body?" This problem must be settled before absolute proof of the true role of the eosinophile, in its relation to histamine in allergy, can definitely be determined.

Therefore, it may be said that as a result of the work by Code in determining the probable function of the eosinophile as a carrier of histamine, the true significance, in the not too far distant future, of eosinophiles in nasal polyps and nasal secretions may give us a better understanding of allergy in rhinology than we have had in the past. It may also explain the recent success with histamine in the treatment of vasomotor rhinitis as recently reported by Hochwald[18] and others from the allergy clinic of this hospital.

In conclusion, I wish to reiterate that this report is presented not as a proof of definite evidence for the true function of the eosinophile as a histamine carrier, but rather a report of evidence that seems highly significant in pointing to such a relationship. It is hoped that when the problem is more fully investigated it may lead to improved treatment of allergic conditions in rhinology.

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VIII

THE FUNCTIONAL ANATOMY OF THE SKULL

THE ANATOMICAL FACTORS IN CRANIO-CEREBRAL INJURIES

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In the head perhaps more than elsewhere, the anatomical factors control the sequence of the pathologic changes so important in deciding between conservative and radical handling. It is also no longer possible to consider the skull as an isolated anatomic unit housing the brain and its coverings and comprised of fixed topographic areas, but rather as a capsule surrounding a fluid organ subject to volume changes induced by the extracranial control of cerebral circulation as well as intracranially induced variations. The protective character of the skull is accomplished at the expense of elasticity and thus these volume changes cannot be readily accommodated by the external bony covering. This failure of the anatomic structures to compensate for fluid volume changes is the basis for most cerebral pathology.

Outside of the cranial cavity, elasticity and resilience of the skull is obtained by air pockets that allow the face and the ear to yield to stress and strain more effectively than solid structures.

The organs of the face in man are also more completely protected than in the lower animals. The outer orbital wall, the zygoma, the alveolus and the palate, as well as the lower jaw form an encasement of the face considerably like the brain encasement, so that volume changes of the orbit are restricted to only the anterior direction. The bony walls inside this peripheral framework can be readily sacrificed to accommodate pathologic volume changes whereas the outer marginal structures must be preserved for functional as well as essential cosmetic effect in faciomaxillary surgery. Only the detailed study of the functional anatomy allows a proper understanding of the varieties of lesions encountered. The weaknesses of anatomic structure preordain the character of the lesions that result from injuries of the various regions. A knowledge of these factors thus allows a better understanding of the clinical pictures.

A comparative study of the male gorilla skull throws light not only on this phase, but on the paranasal sinus development as well.

The system of buttressing the cranial vault against injury in the gorilla is accomplished by a high sagittal ridge which would tend to receive the impact of the blow and then diffuse it laterally to a highly cellular pneumatic petrosal bone which acts as a spongy base and, because of the marked cellularity, serves as a diffusing cushion for the vibrations. In contrast to the ridges, it is surprising to see how thin is the dome of the calvarium.

Posteriorly a horizontal ridge projects around the circumference of the base of the skull extending from one mastoid process to the other. This shelf of bone meets with the sagittal ridge, forming a solid triangle projecting from about 1½ inches to 2 inches in the region of the occipital proturberance.

A third very strong buttress extends from the lateral angular process of the frontal bone along the frontal peak to diffuse the impact of a blow anteriorly into the zygoma and upper jaw. Thus the skull of the male gorilla is buttressed externally. These marked external supports obviate the necessity for a very heavy cranial bone or very marked internal support of the skull. The paranasal sinus area of the gorilla skull is correspondingly extremely pneumatic, offering a degree of resiliency and lightness that makes up for the weight of the jaws and thus allows the whole skull to be relatively light.

In viewing the human skull we find that the construction has shifted so that the protective mechanisms of the skull lie within the skull cavity and the reduced weight of the mandible is counteracted partially by the increased heaviness of the cranial bones themselves. There is also complete disappearance of the frontal peak and the external supports are reduced to a moderately strong, lateral, angular process of the frontal bone.

The skull cap of the human is like that of the gorilla and is composed of three tables, an outer and inner solid table with a layer of diploetic bone serving as a cushion between the two. It is this arrangement which is responsible for the inner table of the calvarium usually fracturing first on impact, whereas the outer table supported by the layer of diploetic bone holds more firmly. In this manner a blow of moderate severity may fracture the inner table of the cranial vault without fracturing the outer table. The distribution of the diploetic layer varies in thickness so that in the region of the squama of the temporal bone it may be extremely thin or absent, thus affording a point of weakness in cranial construction. Similarly the cerebellar fossae may be very thin and subject to fracture.

The dome-like structure of the cranial vault in itself is an important factor in deflecting an impact and causing a wide diffusion of the force. The lines of force radiate into the base of the skull where the various buttresses tend to absorb and diffuse them. In this respect we see a marked difference in the mechanical structure of the human skull with its internal support as compared to that of the gorilla with its external protection.

In the human anterior cranial fossa we see in the midline a free heavy sagittal ridge to which is attached the falx cerebri. This can, in some instances, be quite pronounced and affords considerable cranial support. At its base, where it terminates in the region of the cribriform plate, it is again reinforced by the crista galli, protecting the olfactory bulbs and the cribriform plate, whereas in the gorilla there is virtually no such supporting ridge in the midline along the inner table of the frontal bone and practically no crista galli, the olfactory lobe lying in a deep fossa on the cribriform plate. On either side, the orbital portion of the frontal bone arches high above it. In addition, our anterior cranial fossa is bolstered by the sturdy lesser wings of the sphenoid terminating medially in the anterior clinoid processes where the free ends diffuse the lines of force. Laterally this is accomplished by the external angular processes of the frontal bone.

The weak zone of the anterior cranial fossa is the extremely thin cribriform plate, which consequently is the portion of the anterior cranial fossa most frequently to suffer fracture.

The middle cranial fossa is buttressed on each side by the firm summit of the petrous bone with its apex leading to the posterior clinoid process whose free end again serves to diffuse the lines of force. The weakness of the middle fossa lies in the number of large foramina directly anterior to the petrous portion of the temporal bones. Here the foramen spinosum, foramen ovale, foramen rotundum and foramen lacerum weaken the floor of the middle cranial fossa so that the lines of fracture usually run directly anterior to the petrous bone.

The posterior cranial fossa has as its primary support the internal occipital protuberances as well as the firm bony rim of the foramen magnum. The weaknesses of the floor of the posterior fossa again forms the area customarily fractured.

In viewing the construction of the skull and the direction taken by lines of force following a blow one can consider it as a hemisphere with a flat base and a circular vault. The base is not well suited to receive shock and is weakened by the large foramina and the auditory apparatus, as well as the paranasal air sinuses. The vault, on the

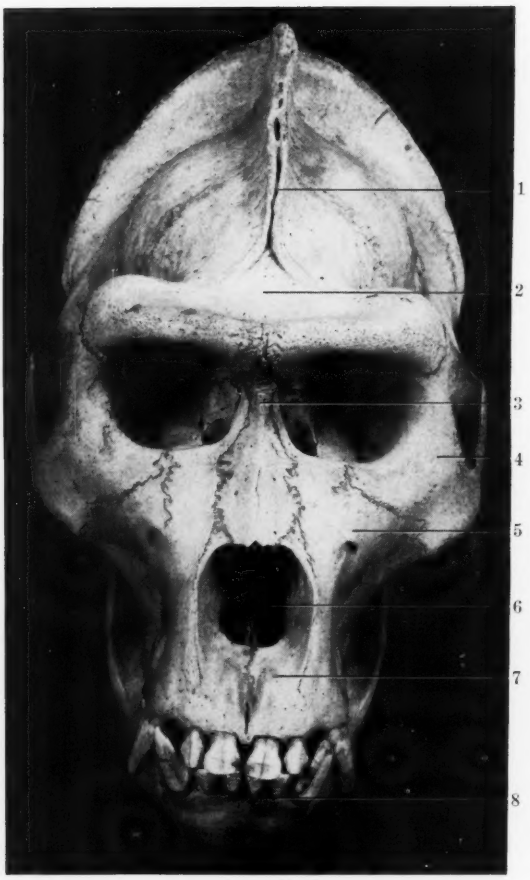

Fig. 1.—Skull of male gorilla: (1) Sagittal crest; (2) Frontal peak; (3) Nasal bone; (4) Malar bone; (5) Maxilla; (6) Nasal Septum; (7) Premaxilla; (8) Mandible.

other hand, is well constructed for glancing off and diffusing blows. The lines of force would be spent as follows:

By dissemination over a large superficial area.

By absorption by the fibrocartilage or fibrous tissue at the synchondrosis or synarthrosis at the basioccipital and petrous areas.

By running along the bony ridges of the base of the skull which terminate blindly. In this manner the lines of force in the frontal region tend to converge on the crista galli. Those from the external angular process of the frontal pass along the lesser wings of the sphenoid to end in the anterior clinoid process. Lines of force from the ear region are projected along the summit of the petrous bone to the apex and to the posterior clinoid process. Blows striking the occipital region travel along the internal occipital crest to the solid margins of the foramen magnum or are diffused outward along the ridges of the lateral sinus. Force passing around the foramen magnum reaches the dorsum sellae and ends in the posterior clinoid process or laterally in the jugular process of the occipital bone where it is absorbed by the fibrous tissue between the process and the temporal bone.

By being passed along any of the blind ends of mere bony ridges to the dura mater which is attached to them.

Finally by travelling in the direction of the pituitary region justly called the water cushion of the brain. There is the tendency for all the lines of force to do this. The clinoid processes bounding this area receive much of the transmitted force and show this by the frequency with which they are torn from their basic attachments.

It was earlier pointed out that the mechanism for the diffusion of force in the male gorilla was external and that the internal supports of the base of the skull were correspondingly diminished. This is particularly evident in respect to the clinoid processes which are reduced to small ridges and to the lack of sharp ridges between the anterior, middle and posterior cranial fossae.

FACTORS IN FRACTURE OF THE BASE OF THE SKULL

The external force may strike the base of the skull directly, as bullet wounds through the mouth or the orbit, a blow on the chin transmitted to the condyle of the mandible or a fall on the feet or buttocks driving the occipital condyles upward. Indirectly, a blow on the vault may be transmitted to the base.

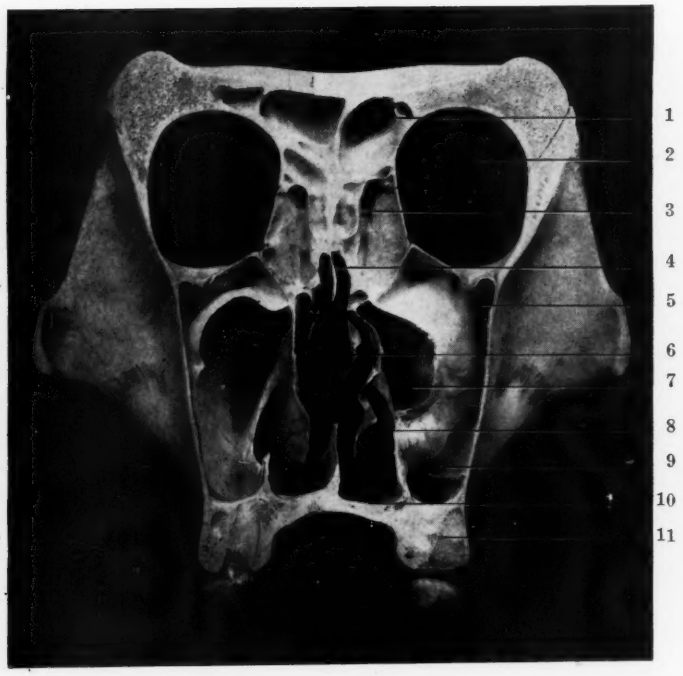

Fig. 2.—Frontal section of skull (male gorilla) I: (1) Frontal sinus; (2) Orbit; (3) Ethmoid sinus; (4) Perpendicular plate of ethmoid; (5) Maxillary sinus primary; (6) Middle turbinate; (7) Maxillary sinus secondary; (8) Inferior turbinate; (9) Alveolar recess; (10) Hard Palate; (11) Alveolar process.

Several theories for this dissemination of force have been advanced. The most common are:

Aran's theory of irradiation that fractures of the base result as extensions from the fractures of the vault, the fracture following the shortest anatomical route to the base. According to Rawling[10] this accounts for about 30 per cent of the cases of basic fractures. He believes, however, that Aran errs in stating that the fracture follows the shortest anatomical route to the base. It is rather influenced by the resistance offered, the weaker areas being selected and the strong buttresses avoided. It is only in the most severe cases that the fracture travels to the base irrespective of the anatomical structure.

In over 60 per cent of Rawling's cases of fracture of the base, the injury was received over one of the following regions: anteriorly at the frontal eminence or supra-orbital ridges; anterolaterally over the external angular frontal process; laterally over the temporo-auricular and mastoid regions; posteriorly over the superior curved line of the occipital bone or over the external occipital proturberance. In all of these cases the blow struck near the base of the skull and the resultant fracture was by direct violence. The fracture travelled the base and split it like a chisel splits a board of wood.

The contre coup theory which Rawling rejects has been advanced to explain those cases in which there is a fracture of the base following a blow on the vault with no fracture of the vault itself. The theory is that from the point struck a wave is transmitted through the semifluid brain producing a fracture at some more distant point. This was conceived to explain such instances as isolated fractures of the orbital roof. Rawling, although conceding that waves are transmitted through the brain and cerebrospinal fluid, does not believe them capable of producing a basic fracture. He feels rather that a blow on the vault may fail to produce a local lesion; yet when the lines of force are transmitted to a weaker area in the base, they may cause a fracture in such delicate structures as the cribriform plate or the thin orbital plate.

The bursting and compression theories regard the skull as a highly elastic sphere, compression of which leads to a diminution in the diameter along the axis of greatest pressure with corresponding bulging in the other diameters. When the bulging exceeds the limits of elasticity, the fracture runs parallel to the line of force and the bone bursts open along the convexity producing a bursting fracture. When the line of fracture is at right angles to the direction of the compressing force, a compression fracture results. The elasticity

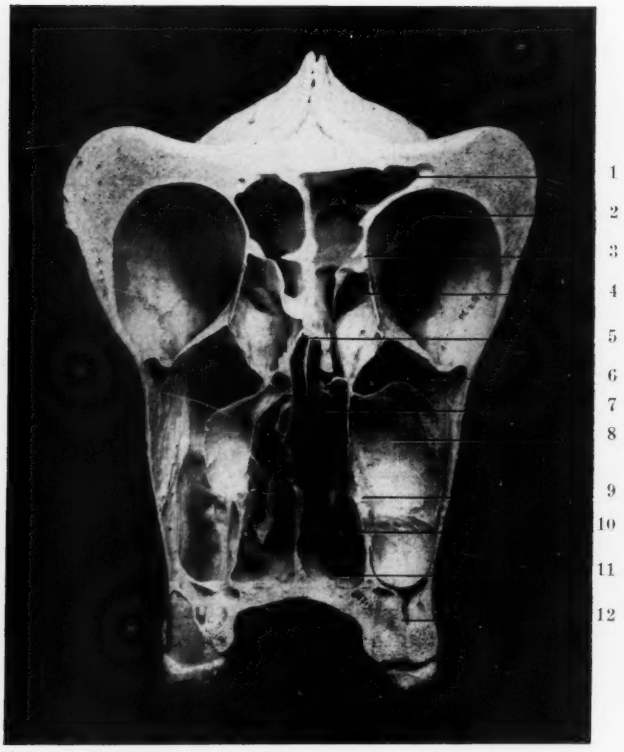

Fig. 3.—Frontal section of skull (male gorilla) II: (1) Frontal sinus; (2) Orbit; (3) Lamina papyracea; (4) Ethmoid sinus; (5) Perpendicular plate of ethmoid; (6) Maxillary sinus primary; (7) Middle turbinate; (8) Maxillary sinus secondary; (9) Ostium inferior turbinate; (10) Inferior turbinate; (11) Floor of nose; (12) Alveolar process.

of the skull is no doubt less than this theory assumes. Rawling states that about five per cent of his cases afforded examples of bursting and compression fractures. These result from bilateral compression, as from falls on to the buttocks and from blows applied to the angle of the jaw.

Aside from the theories of fracture the anatomical construction of the base of the skull presents several outstanding factors. The skull may be viewed as composed of two halves, one lying anterior to the occipital condyles, the other posterior to and including the condyles with their vertebral attachments. A glance at the base of the skull as seen from below shows a weak line including the petro-tympanic fissure, the petrosphenoidal sutures, both foramina lacera media and the sphenoidal sinus between them. The two halves appear practically cemented together by the union of the basisphenoidalis to the basi-occipitalis. Thus a blow struck on the anterolateral region of the head tends to split off the anterior segment from the more fixed posterior portion with the fracture following the weak line described. This conception is further supported by the fact that the greater number of middle fossa fractures follow this line and it may be considered as the typical fracture of the base of the skull.

Rawling, as a result of analysis of his cases, has classified as follows the anatomic structures fractured by blows in the various regions of the skull, bearing in mind that exceptions due to overwhelming force may occur.

When the force strikes the median frontal region, the fracture passes backwards from the perpendicular plate of the frontal bone to the cribriform plate of the ethmoid, thence between the optic foramina to the body of the sphenoid. The thin sinus roof is usually comminuted. From there the fracture diverges to the opposite side, and tearing off the posterior clinoid process, passes along the petro-occipital suture to the jugular foramen, being then continued on the other side of that foramen along the masto-occipital suture, and so again to the vault.

In a blow hitting the lateral frontal region at the external angular frontal process, the fracture passes across the anterior fossa towards the sphenoidal fissure, tearing away the anterior clinoid process, and again comminutes the roof of the sphenoidal sinus. Progressing onwards, with or without fracturing the posterior clinoid process, the fracture passes either along the anterior part of the petrous bone at its junction with the greater wing of the sphenoid towards the opposite middle and external ears, or along the petro-

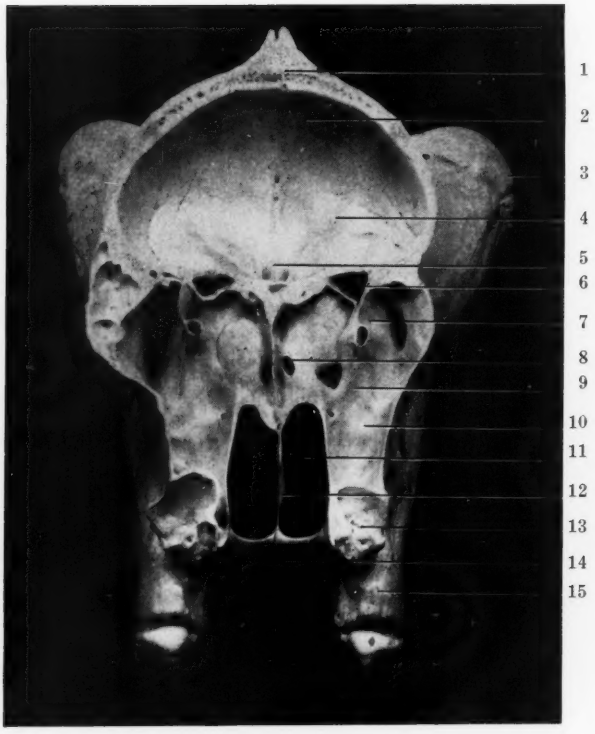

Fig. 4.—Frontal section of skull (male gorilla) III: (1) Sagittal crest; (2) Frontal bone; (3) Malar prominence; (4) Anterior cranial fossa; (5) Olfactory fossa; (6) Superior orbital fissure; (7) Canal for maxillary nerve; (8) Ostium sphenoidale; (9) Sphenoid sinus; (10) Pterygoid process; (11) Choana; (12) Nasal septum; (13) Pterygoid process; (14) Hard palate; (15) Alveolar process.

occipital suture to the jugular foramen, and continues along the masto-occipital suture as in the previous case.

In a force striking the region of the external ear, the fracture passes across the roof of the bony auditory meatus toward the junction of the anterior and inner walls of the middle ear, the membrane undergoing a variable amount of destruction and displacement. The fracture is then continued across the tegmen tympani, and after following the petrosphenoidal suture reaches the foramen lacerum medium, being again continued on the opposite side of that foramen to the sphenoidal body. Thence it pursues one of two courses. Most commonly the fracture passes backwards obliquely to the opposite middle and external ears, following a course similar to that already indicated.

In such cases the fracture may extend on each side up on to the vault in such a manner that the two segments are merely united by the soft parts; whether the fracture be so complete or not, a more minute examination of the line of separation will show many interesting points. An inspection of the anterior aspect of the posterior fragment shows that the fracture passes just in front of the geniculate ganglion of the facial nerve, the ganglion being laid bare, while its petrosal branches are usually torn. The fracture usually passes anterior to the eustachian tube and the horizontal part of the internal carotid artery. On examining this posterior fragment the following structures will be seen, passing from without inwards: the posterior half of the external auditory meatus, the mastoid antrum, the lacerated membrane and the ossicles of the middle ear, the geniculate ganglion of the facial nerve, the eustachian tube, the horizontal part of the internal carotid artery, the gasserian ganglion, and the posterior half of the sphenoidal sinus in the midline.

After reaching the sphenoidal body, the alternative course for the fracture to pursue is to pass toward the opposite sphenoidal fissure and, tearing off the anterior clinoid process, to cross the anterior fossa parallel to the original direction but not in the same straight line.

When the force is applied to the mastoid region, the fracture follows the occipitomastoid suture to the jugular foramen, and is again continued on the opposite side of that foramen along the petro-occipital suture toward the apex of the petrous bone. It then passes across the sphenoidal body to the sphenoidal fissure of the opposite side, and so across the anterior fossa. It is especially common in this particular variety of fracture to find fissures diverging from the region of the sphenoidal sinus forwards toward the cribriform plate of

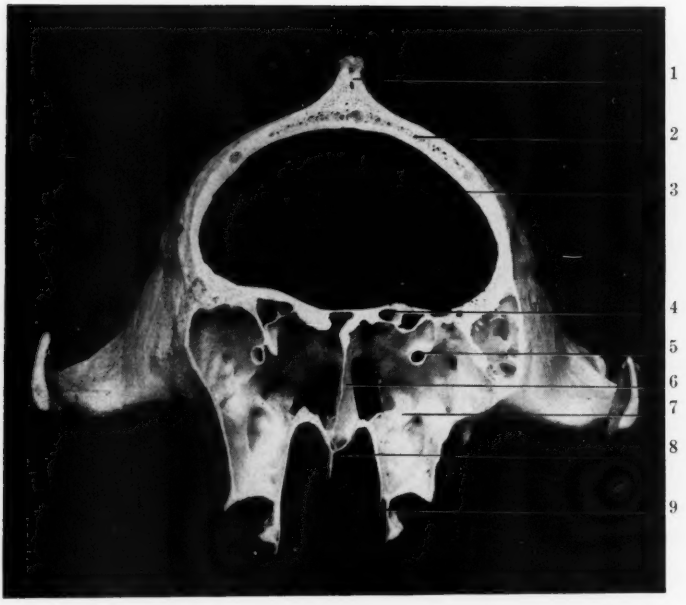

Fig. 5.—Frontal section of skull (male gorilla) IV: (1) Sagittal crest; (2) Parietal bone; (3) Middle cranial fossa; (4) Optic canal; (5) Maxillary canal; (6) Septum sphenoidale; (7) Sphenoid sinus; (8) Nasal septum; (9) Pterygoid process.

the ethmoid, these fissures usually passing between the optic foramina.

This fracture is also peculiar in so far as, when the degree of separation along the occipitomastoid suture is excessive, there is special danger of tearing the lateral sinus wall where the sinus begins to turn downwards and inwards.

In the case of force striking the lateral occipital region, the fracture passes across the cerebellar fossa and strikes the foramen magnum immediately behind the condyle. Starting again from a similar point on the opposite side of the foramen, the fracture passes outward to the jugular foramen. Again, two courses are now available, the fracture either cutting outward across the body of the petrous, "external" to the internal auditory meatus and cutting across the facial nerve in the region of the geniculate ganglion, and finally terminating in the roof of the middle ear; or else passing along the petro-occipital suture and so to the foramen lacerum medium, the sphenoidal fissure, and the anterior fossa as in the previous case.

In a blow on the posterior occipital region, the resultant fracture may vary according to the direction of the applied force. It it strikes the posterior occipital region at right angles to the transverse axis of the skull, there follows a fracture which, on reaching the posterior margin of the foramen magnum, is continued again on the opposite side of the foramen along the dorsum sellae. When the force is more oblique in direction, as is usually the case, the fracture traverses the thin cerebellar fossa to the outer margin of the jugular foramen, and then follows one of the two courses indicated in the previous case. More commonly the fracture cuts across the petrous bone.

The symptoms resulting from fracture of the skull represent primarily the effects of the trauma on the brain and its vascular mechanism. The protection by the water cushion of the brain formed by the cisternae and the cerebrospinal fluid distribution around the pituitary do much to minimize injury to the vital centers. The skull fracture is a secondary consideration and may manifest itself by evidence of hemorrhage, escape of cerebrospinal fluid, extrusion of brain matter, passage of air from the air sinuses into the surrounding tissues and involvement of the cranial nerves. Characteristic lesions can be classified according to their location in the anterior, middle and posterior cranial fossae and are so grouped by Rawling.

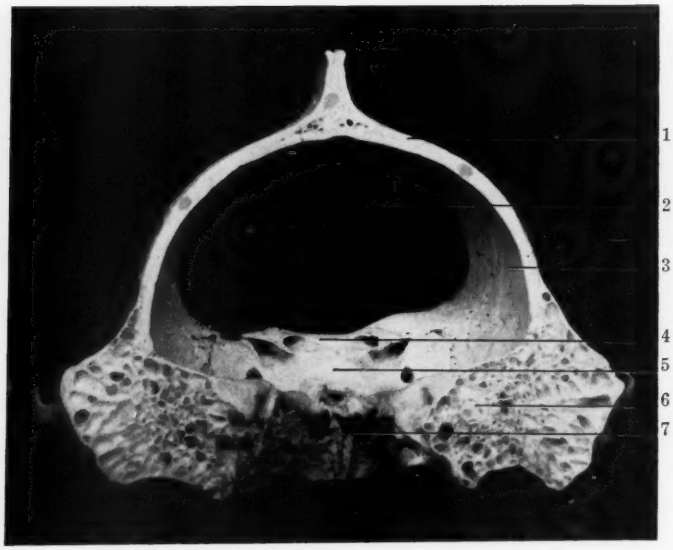

Fig. 6.—Frontal section of skull (male gorilla) V: (1) Parietal bone; (2) Middle cranial fossa; (3) Temporal bone; (4) Sulcus chiasmaticus; (5) Pituitary fossa; (6) Labyrinth; (7) Sphenoid sinus.

SYMPTOMS INDICATING FRACTURE OF THE ANTERIOR FOSSA

Subconjunctival hemorrhage usually makes its appearance at the outer canthus of the eye, progressing inward toward the corneoscleral margin, and in the more severe cases completely surrounding the cornea, bulging the conjunctiva forward in such a manner as to constrict the field of vision. The extravasated blood is usually bright red in color, makes its appearance within a few hours of the accident, and reaches its maximum within 36 to 48 hours.

In some cases a condition of subconjunctival edema (chemosis) is observed. This also usually originates at the outer canthus.

Taken by themselves, neither hemorrhage nor edema are of any great diagnostic value. Both conditions, however, aid materially in confirming the diagnosis.

The blood is almost invariably completely absorbed, and no ill effects remain.

Palpebral and peripalpebral hemorrhage is seen in most cases of fracture of the anterior fossa. This form of hemorrhage differs from the one mentioned above in that it usually commences at the inner canthus of the eye, then progresses in the outward direction. The extravasated blood may be wholly anterior to the suspensory ligaments of the lid, in which case it may be surmised that the fracture only involves the squama of the frontal bone. More commonly however, the cribriform plate of the ethmoid shares in the lesion, in which case palpebral, peripalpebral, and subconjunctival hemorrhage are all present.

Orbital hemorrhages may be so extensive that marked forward protrusion of the globe exists. The time at which proptosis makes its appearance and the degree to which it progresses vary according to the nature of the lesion. Severe proptosis appearing almost at once implies a fracture associated with injury to the cavernous sinus or to the internal carotid artery. Moderate proptosis appearing after a few hours, would indicate a fracture involving the walls of the orbit, the blood being derived from lacerated ethmoidal and other small vessels. If it appears days or weeks after the accident and is progressive, it suggests a fracture involving the region of the sphenoidal body and complicated by the formation of a fistulous communication between the cavernous sinus and the carotid artery, arteriovenous aneurysm.

Retinal hemorrhages were reported by Fleming, in 1902, in 12 cases of fracture of the skull, all except one being fractures of the

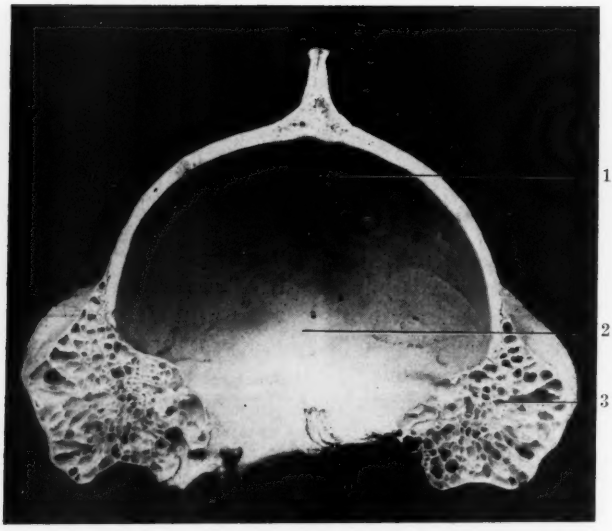

Fig. 7.—Frontal section of skull (male gorilla) VI: (1) Occipital bone; (2) Posterior cranial fossa; (3) Temporal bone.

base. All cases were associated with hemorrhage into the subarachnoid space, and when this hemorrhage was of a unilateral nature the retinal changes were likewise homolateral. It was also found that in four cases of cerebral hemorrhage without osseous lesion retinal hemorrhages were present in three, these three being all associated with considerable effusion into the subarachnoid space.

These observations are not only of value in the general diagnosis of intracranial lesions, but are also of considerable importance in the differential diagnosis between extra- and intra-dural hemorrhages.

Hemorrhage from the nose and mouth is almost invariably present in fractures of the anterior fossa, with the inference that the fracture involves the cribriform plate. The anterior and posterior ethmoidal arteries run in a groove-like channel from the orbit medially to the cribriform plate. The vessels hug the bone which forms simultaneously the roof of the ethmoid sinus and the floor of the anterior cranial fossa. A fracture therefore easily tears the ethmoidal vessels. This is the usual source of the blood which flows from the nose or passes backwards into the pharynx to be swallowed and vomited up later, or flows from the mouth.

Escape of cerebrospinal fluid occurs when the fracture involves the cribriform plate of the ethmoid with laceration of the underlying dura mater and arachnoid and the prolongations of those membranes along the olfactory nerves. Increase in the flow of this clear fluid from the nose or throat on assuming the recumbent position also is suggestive of cerebrospinal rhinorrhea. The semirecumbent position is advisable in these cases as well as the strict avoidance of sneezing, blowing the nose, coughing or straining. The escape of clear fluid from the ear, through the eustachian tube into the throat and nose, could also be an indication of an opening of the membranous labyrinth. Cerebrospinal fluid collected early shows low chlorides, little or no albumin and the presence of a reducing sugar, whereas mucus of nasal origin is high in protein and chlorides and gives no reducing reaction.

Blood contamination of the cerebrospinal fluid may obscure these tests and if it is collected late may be further masked by serous exudate. It may be clinically considered cerebrospinal fluid if it begins within 24 hours after the trauma, is colorless, fairly profuse and continues for two or three days or longer. The quantity can vary from slight amounts to large amounts. I recall seeing a case of escape of cerebrospinal fluid from the inner ear that required changing of a

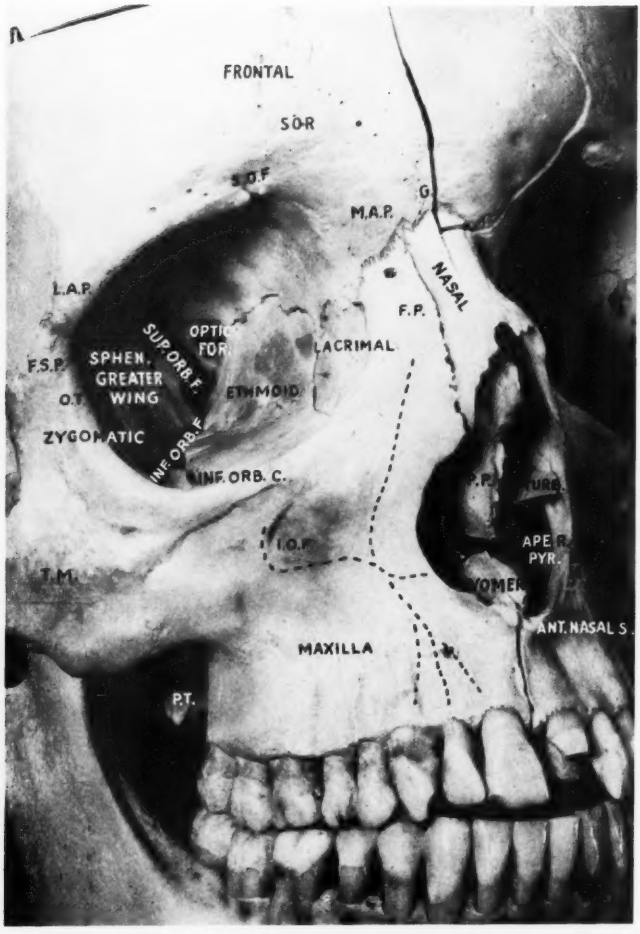

Fig. 8.—The skeletal structure of the face: S.O.R., Supra-orbital ridge; S.O.F., Supra-orbital foramen; L.A.P., Lateral angular process of frontal bone; F.S.P., Frontosphenoidal process of zygomatic bone; O.T., orbital tubercle of zygomatic bone; T.M., Malar tuberosity; I.O.F., Infra-orbital foramen; P.P., Perpendicular plate of ethmoid; F.P., Frontal process of maxilla; M.A.P., Medial angular process of frontal bone; G., Glabella; ANT. NASAL S., Anterior nasal spine; The broken line indicates the small bony canal for the anterior superior alveolar nerve and artery. The ascending canal is for an arterial twig emerging at the sutura notha to anastomose with the facial. The short horizontal canal is for nerve and arterial twigs to the nasal mucosa and opens on the nasal aspect of the frontal process of the maxilla.

mastoid dressing several times daily; at each time the large dressings were completely drenched.

The prognosis in cases of cerebrospinal rhinorrhea is considered generally favorable with surgical intervention, providing it is warranted by the patient's general condition. As in other cranial injuries, recovery from shock should precede surgical intervention. Early operation is particularly indicated when the fracture is over the frontal sinus and extends to the inner sinus wall. When the anterior cranial floor is affected, a short period of conservative therapy can be carried out. A fistulous tract is a potential menace of meningitis and should be eradicated surgically. Resection of the tract and closure of the dura by transplant gives a rapid and safe solution to this problem. Syringing of the nose or ear, or packing obstructing the outflow of the cerebrospinal fluid is absolutely contraindicated. For the ear, external dressings may be used and the dressings changed as soon as they are soaked. The ear canal can be wiped out and sulfanilamide ascorbate lightly insufflated. No packing is put into the canal.

Escape of air into the surrounding tissues may result when the fracture line runs through the paranasal sinuses or the mastoid and leads to pneumocephalus. Air may escape into the subdural space, the arachnoid sulci and the cisternae and into the ventricles or brain substance giving rise to pneumocephalus recognizable by x-ray examination. Air may also find its way into the pericranial tissue giving rise to surgical emphysema. There is probably some degree of pneumocephalus in most cases of cerebrospinal rhinorrhea. This would be aggravated by sneezing, coughing or straining.

Involvement of the cranial nerves. In the anterior cranial fossa we find three groups that may be affected. They are the olfactory, the optic and the nerves passing through the superior orbital fissure, the third and fourth, and the ophthalmic division of the fifth and sixth.

The Olfactory Nerve. The great majority of anterior fossa fractures traverse the cribriform plate, necessarily injuring the fine branches of the olfactory bulb. The bulb itself may be lacerated, with or without injury to the under surface of the frontal lobes. The olfactory bulbs lie at a lower level than the frontal lobes and occupy the depression formed by the weak cribriform plate. It is not unnatural, therefore, for injury of the olfactory bulb to occur readily in median fracture of the anterior cranial fossa. Anosmia, whether uni-or bilateral, transient or permanent, generally results from direct

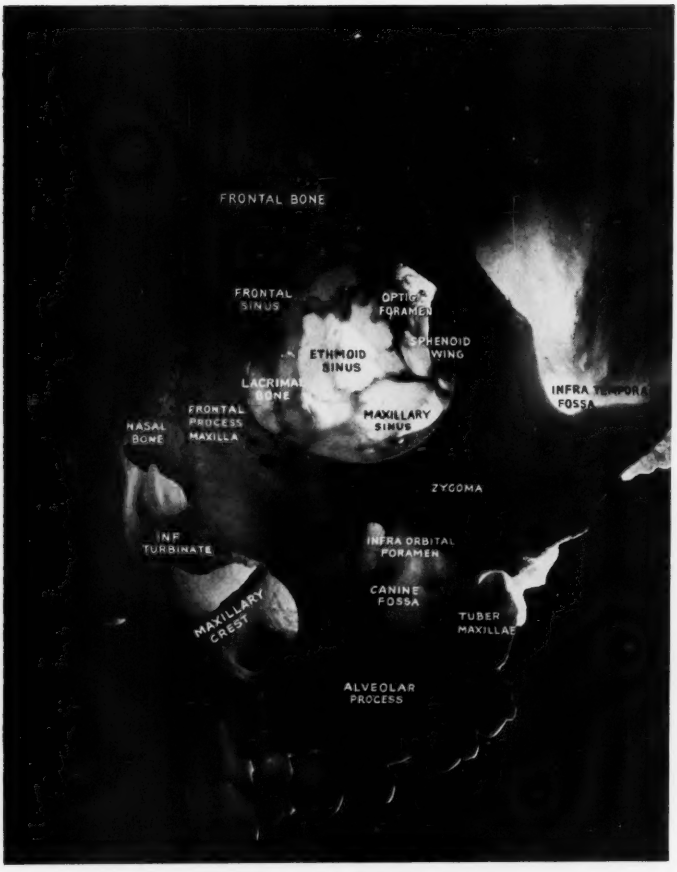

Fig. 9.—Transillumination of skull to show the relationship of the paranasal sinuses to the orbit.

injuries of the cribriform plate with associated lacerations of the olfactory nerves. These lesions are not necessarily associated with cerebrospinal rhinorrhea. It is difficult to estimate the presence, or the degree of immediate loss, of smell because of the general condition of the patient and because the nostrils are usually more or less filled with blood coagulum. Experience shows, however, that early loss of smell is the rule and total and permanent anosmia the exception. Anosmia is usually associated with some degree of loss of taste. Partial recovery of the sense of smell is commonly observed.

The Optic Nerve. Many cases have been recorded in which visual defects resulted from blows to the head. Blindness may be partial or complete, immediate in onset or developing at some future date. In the latter case, the loss of vision is due to retinal changes or results from post-neuritic atrophy.

The occurrence of complete or partial blindness as the immediate result of injury is, at first sight, difficult to explain, for the vast majority of anterior fossa fractures avoid the immediate vicinity of the optic foramina, passing by preference between the two foramina or diverging toward the sphenoidal fissures. Small fissured fractures not infrequently radiate through the optic foramina, usually however, of so slight a nature as to be incapable of leading to any gross lesion of the optic nerves. Hemorrhage into the sheath of the nerve is probably responsible for a certain proportion of the cases, more especially those in which there is a peripheral concentric loss of vision, the more central fibers escaping. It is possible, also, that cases evidencing temporal or nasal blindness may be due to a contre coup contusion of the nerve through its being forcibly driven against the bony boundaries of the foramen. Taking into consideration however the very frequent presence of a fracture through the anterior clinoid process and the usual displacement of that process, it would appear probable that immediate and more or less complete loss of vision results from the compression and crushing of the optic nerve by reason of the pressure exercised by a displaced clinoid process. Stereoscopic x-ray studies of the clinoids may be helpful in establishing this diagnosis.

The prognosis varies according to the cause of blindness. When resulting from concussion of the nerve trunk or from hemorrhage into the sheat, certain fibers may regain their function. In the majority of cases, however, blindness is immediate and permanent.

The Nerves Passing Through the Superior Orbital Fissure. The ophthalmic division of the fifth nerve is rarely injured to such a de-

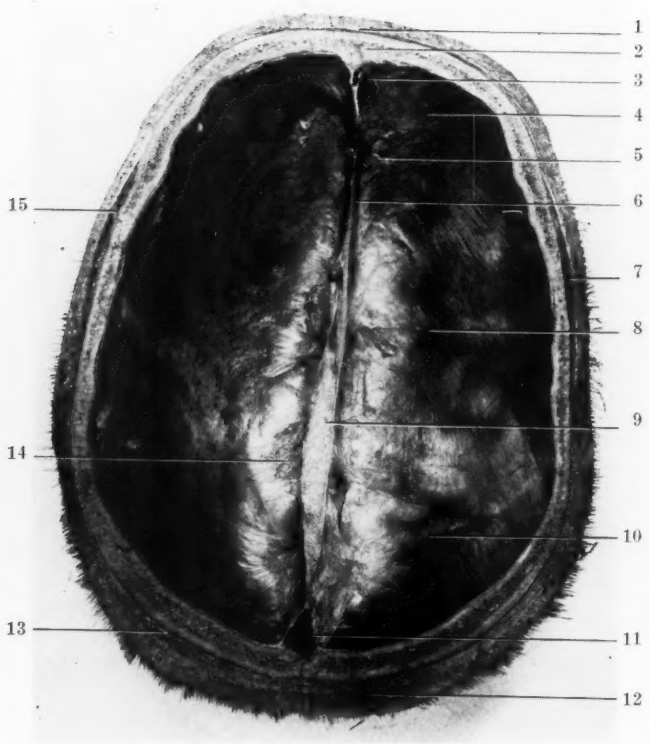

Fig. 10.—The calvarium with dura mater attached: (1) Galea aponeurotica; (2) Frontal bone; (3) Sagittal sinus; (4) Anterior cranial fossa; (5) Pacchionian body; (6) Falx cerebri; (7) Temporal muscle; (8) Middle cranial fossa; (9) Sagittal sinus; (10) Posterior cranial fossa; (11) Sagittal sinus; (12) Galea aponeurotica; (13) Occipital bone; (14) Lacuna lateralis sagittal sinus; (15) Temporal muscle.

gree as to cause anesthesia of all the regions supplied. Blood extravasation into the surrounding regions or direct involvement of one of the branches of the nerve often results in areas of anesthesia, and few cases have been recorded in which there was complete anesthesia of both cornea and conjunctiva, with subsequent ulceration and sloughing. The ethmoidal nerve may be implicated as the result of a fracture involving the cribriform plate, while the supra-orbital and the supratrochlear branches may be damaged by fractures of the squama of the frontal bone.

The third nerve is similarly subject to injury, in any part of its orbital course. It is quite exceptional, however, for the whole trunk to be affected, some of the branches being taken, others left. The fourth nerve is also occasionally involved, generally in association with other orbital nerves such as the sixth.

When anesthesia or paralysis of muscles results from pressure exercised on the nerves by extravasated blood, the ultimate prognosis is not unfavorable. When due to direct implication, in the line of the fracture, the prognosis is much more uncertain, partial or complete loss of function resulting.

SYMPTOMS INDICATING FRACTURE OF THE MIDDLE FOSSA

Hemorrhage into the temporal region. The extravasated blood may either be confined to the temporal region (temporal hematoma) or diffused throughout the subaponeurotic space. A temporal hematoma is always highly suggestive of a fracture involving the temporal fossa, especially in the event of marked outward bulging with stretching and discoloration of the overlying tissues. In many cases also the hematoma pulsates, in which case it can be presumed that fracture of the temporal fossa is associated with hemorrhage from a lacerated middle meningeal artery. In such cases the application of pressure to the hematoma may lead to the development of fits on the contralateral side, originating in the face or the arm regions and spreading to the higher cortical motor area.

Hemorrhage from the ear and mouth. The great majority of middle fossa fractures involve the external auditory meatus, passing inward across the roof and floor of the middle ear toward the body of the sphenoid. Examination will show that the fracture passes inward toward the junction of the inner and anterior walls of the middle ear, that is to say, toward the tympanic orifice of the eustachian tube. The membrana tympani undergoes a variable degree of destruction. In lesser cases the membrane is torn in its upper and anterior part only—in the region of Shrapnell's membrane; while in the more seri-

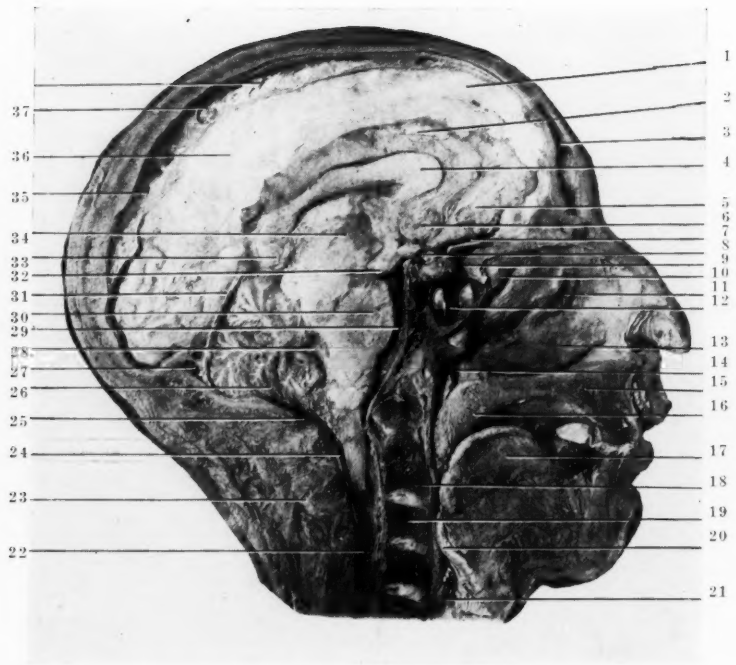

Fig. 11.—Ventricular and subarachnoid spaces viewed in median sagittal section of the head: (1) Falx cerebri; (2) Gyrus cinguli; (3) Frontal sinus; (4) Genu corporis callosi; (5) Frontal lobe; (6) Hypothalamic area; (7) Cisterna interpeduncularis; (8) Pituitary; (9) Septum sphenoidalis; (10) Superior turbinate; (11) Middle turbinate; (12) Sphenoid sinus; (13) Inferior turbinate; (14) Eustachian tube; (15) Hard palate; (16) Soft palate; (17) tongue; (18) Epistropheus; (19) Third cervical vetebra; (20) Epiglottis; (21) Arytenoids; (22) Spinal cord; (23) Posterior cervical muscles; (24) Cisterna cerebello medullaris; (25) Foramen magendi; (26) Cerebellum; (27) Transverse sinus; (28) Fourth ventricle; (29) Cisterna pontis; (30) Pons; (31) Tentorium cerebelli; (32) Hypothalamus; (33) Choroid plexus; (34) Third ventricle and thalamus; (35) Superior sagittal sinus; (36) Pacchionian bodies (37) Sagittal sinus wall.

ous cases it may be completely destroyed. The blood that escapes from the ear is derived from those vessels that supply the lining of the external canal and the middle ear, from the numerous tympanic vessels, from the lateral sinus, and from the middle meningeal artery.

The amount of blood which escapes varies according to the source of the hemorrhage. When the hemorrhage occurs from the smaller vessels, the blood either clots in the external meatus or trickles from the ear. In the more severe cases the hemorrhage is profuse and long continued. In some instances the dome of the jugular bulb reaches up into the floor of the tympanum and when lacerated gives rise to profuse bleeding from the middle ear. In simple fracture of the tegmen tympani, the bleeding from the ear may be slight, and inspection several days later shows a dry blood crust adherent to the upper anterior portion of the external auditory canal close to the drum membrane. In medicolegal cases, this may be the only demonstrable evidence of a small linear fracture.

The possibility of an extradural hemorrhage developing must be borne in mind. Since laceration of the middle meningeal artery may occur in about 30 per cent of the cases of middle fossa fractures, bleeding from both ears would indicate a typical basic fracture and grave prognosis. It must nevertheless be remembered that simple rupture of the drum membrane with hemorrhage and no fracture is a common occurrence. Evidence of cerebral injury usually accompanies fracture.

Hemorrhage from the mouth is slight when from the sphenoid or the pharyngeal vessels, or profuse when from the cavernous sinus or the carotid artery. The latter would occur with shattering of the body of the sphenoid.

The escape of cerebrospinal fluid from the ear in middle fossa fractures is rare since the typical fracture usually passes anterior to the genu of the facial nerve and thus does not affect the arachnoid prolongations of that nerve. However, in occipital fractures running anteriorly, the line runs through the petrous bone at right angles so as to cut the facial nerve at right angles and expose the arachnoid in the region of the genu. This would be the usual fracture associated with the escape of cerebrospinal fluid from the external auditory meatus. Rarely, in this type, does the cerebrospinal fluid pour into the pharynx through the eustachian tube in the presence of an in tact drum membrane. Extensive fracture of the basisphenoidalis and the basi-occipitalis may allow cerebrospinal fluid to escape from the cisterna basilis.

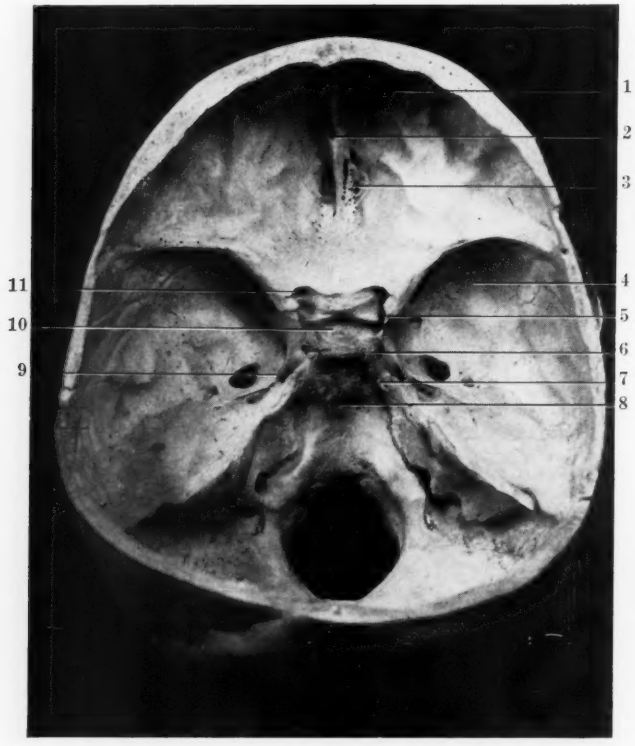

Fig. 12.—Base of skull showing varying thickness of walls: (1) Anterior cranial fossa; (2) Foramen calcium; (3) Cribriform plate; (4) Middle cranial fossa; (5) Anterior clinoid process; (6) Posterior clinoid process; (7) Apex of petrous bone; (8) Basi occipitalis; (9) Carotid canal; (10) Pituitary fossa; (11) Optic canal.

Escape of brain matter from the ear and the nose should have the same treatment as described for hemorrhage. The tissue is wiped away and protected by sterile loose dressing. Operative measures are indicated when the symptoms point to compression.

Involvement of Nerves. The second and third divisions of the fifth nerve pass respectively through the foramen rotundum and the foramen ovale. Both lie anterior to the petrosphenoidal suture, traversed by the majority of the middle fossa fractures. These two nerves are therefore seldom involved.

In certain rare instances, a fracture, passing in the anteroposterior direction, may cut across the apex of the petrous bone in close relation to the fossa for the gasserian ganglion, in which case all three terminal divisions of the fifth nerve may suffer.

The sixth nerve may be involved either by itself or in conjunction with other cranial nerves. In the latter case the paresis is due to blood extravasated in the superior orbital fissure or in the orbital cavity. In the former case the nerve is injured where it grooves the lateral aspect of the dorsum sellae, a process frequently fractured in lesions of the middle fossa or compressed as it passes through the canal of Dorello. Fractures tend to pass obliquely across this process, one nerve usually escaping. The prognosis as to functional recovery is very uncertain.

There can be no doubt that the seventh nerve on account of its complicated intrapetrous course is more frequently involved than any other cranial nerve.

The question of facial nerve implication is so intimately associated with involvement of the eighth nerve that the two must be considered together. Thus, the cases may be classified as follows: those cases of paresis of the facial nerve with a variable degree of deafness and those cases of complete facial paralysis with complete deafness.

The greater number of middle fossa fractures involve the middle and external ears, as is evidenced amongst other symptoms by hemorrhage from the ear. Some degree of facial paralysis is frequently existent, not always evident at first sight, but requiring careful examination and comparison between the two sides of the face. The fracture involves both the roof and the floor of the external ear and passes inward toward the junction of the anterior and inner walls of

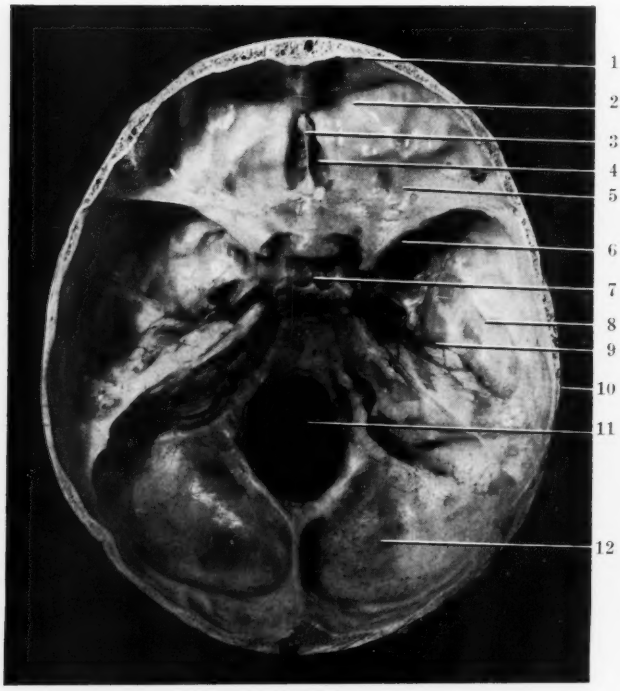

Fig. 13.—Base of skull showing natural line of fracture of middle fossa: (1) Squama of frontal bone; (2) Orbital plate of frontal; (3) Crista galli; (4) Cribriform plate; (5) Anterior cranial fossa; (6) Lesser wing of sphenoid; (7) Pituitary fossa; (8) Middle cranial fossa; (9) Natural line of fracture; (10) Squama of temporal bone; (11) Foramen magnum; (12) Posterior cranial fossa.

Fig. 8 shows line of "natural fracture."

the middle ear; the membrana tympani undergoes a variable degree of destruction and the ossicles may be injured. The fracture then passes inward toward the petrosphenoidal suture in such a manner that the geniculate ganglion of the facial nerve is exposed and laid bare on the anterior aspect of the posterior portion of the skull. The greater superficial petrosal nerve, which leaves the geniculate ganglion and passes anteriorly to form part of the vidian nerve root of the sphenopalatine ganglion, carries taste fibers to the side of the mouth and the tip of the tongue. These taste disturbances are commonly associated with facial nerve paralysis.

The facial nerve, therefore, escapes direct injury except in so far as the ganglion may be compressed by blood-clot or fragments of bone. Partial loss of function results. In most cases the blood is absorbed and a complete recovery may be anticipated. The degree of deafness is directly proportionate to the damage incurred by the membrana tympani and the ossicles. The degree of hearing loss may be slight and apparently out of proportion to the severity of the injury.

Functional testing of the labyrinth in these cases may also show a normal reaction to rotation and caloric testing with no disturbance in falling or pass pointing and no spontaneous nystagmus. The medicolegal aspects may be important in this type of case since total and permanent loss of hearing is usually claimed. I recall the case of a 45-year-old man who fell down a flight of stairs and was reported to have sustained a simple fracture of the right frontal bone extending for about 2 cm. upward from the orbital rim at the head of the eyebrow. The patient suffered a temporary loss of vision, diminution in the sense of smell and taste, right facial paralysis and loss of hearing on the right side. Functional testing of the labyrinth, three months later, showed normal reactions to rotation and caloric testing and progressive improvement of hearing with partial recovery of the facial palsy. On the basis of the anatomical findings, a fracture of the anterior and middle fossae was postulated. The probable course of the fracture was across the anterior cranial floor, tearing off the anterior clinoid process, and extending through the middle fossa to the outer third of the petrous bone in the region of the tegmen antri. Subsequent stereoscopic x-ray films confirmed this course. The fracture had crossed the greater superficial petrosal nerve and the geniculate ganglion with hemorrhage into the antrum of the mastoid and failed to strike the labyrinth.

In the second group of cases a different picture is obtained. Usually it is the result of blows applied to the occipital region. The

fracture traverses the thin cerebellar fossa toward the outer angle of the jugular foramen, then cuts across the petrous bone, external to the internal auditory meatus, and terminates, usually by comminution, in the tegmen tympani. It is in the transpetrous part of the fracture that the damage is done, for not only is the facial nerve cut across in the region of the ganglion, but the auditory apparatus is severed into two parts.

In this class of fracture, though facial paralysis and deafness are both immediate in onset and permanent in duration, there is, in many cases, no bleeding from the ear as the membrana tympani may be uninjured.

The history of bleeding from the middle ear is usually considered so important in the diagnosis of ear injury secondary to skull fracture that in the absence of bleeding the severity of the injury may be underestimated or not observed till the patient has recovered from other manifestations of brain injury. He may then notice his loss of hearing. However, in these cases functional testing of the labyrinth will establish the loss of the inner ear. In late cases this may be made difficult by compensatory reaction to rotation from the opposite ear. The symptoms of cerebral injury, such as nausea, vomiting, and headache, may also mask the vestibular irritation at the onset unless nystagmus is observed. Bilateral injury of this type is exceedingly rare.

Fracture of the mastoid may occur with facial paralysis. Complete facial paralysis may be associated with the following symptoms: epiphora, conjunctivitis, and keratitis from paralysis of the obicularis palpebrarum; loss of taste from involvement of the chorda tympani and the geniculate ganglion; impaired nasal air-entry into the ear from paralysis of the stapedius muscle; impaired mastication from involvement of the buccinator muscle; impaired secretion of saliva from cutting off the secretory and vasodilator fibers of the chorda tympani.

Lastly, it is necessary to add that facial paralysis developing some days or weeks after the accident, though sometimes dependent on degeneration of nerve fibers as the result of pressure in the region of the geniculate ganglion, may also arise from an ascending neuritis or from meningeal infection.

SYMPTOMS INDICATING A FRACTURE OF THE POSTERIOR FOSSA

External hemorrhage. In fractures of the posterior fossa, blood effused into the deeper tissues of the scalp has considerable difficulty in coming to the surface and thus making itself evident. Further-

more, the resistance offered by the nuchal muscles tends to confine the blood to the subtentorial region, thus adding to the already grave prognosis of fractures in this region. On careful palpation, however, it will be noted that the nuchal tissues present a doughy or boggy condition, while ecchymosis becomes evident after 24 to 36 hours. A peculiar ecchymotic patch is occasionally observed appearing in front of the mastoid process and travelling upward in a curved direction, with the concavity forward following the outline of the ear. It is said to result from tracking of blood along the course of the posterior auricular artery. Whether this is the case or not, the hemorrhage usually implies a separation along the line of the mastooccipital suture. Neither escape of cerebrospinal fluid nor escape of brain matter is present.

Involvement of nerves. When dealing with fractures of the middle fossa, reference was made to the implication of the seventh and eighth pairs of nerves as the result of a fracture traversing the posterior fossa of the skull toward the outer angle of the jugular foramen and cutting across the petrous bone.

The ninth, tenth, and eleventh cranial nerves may be injured in the same variety of fracture. These three nerves are, however, so protected by their dural sheaths that they generally escape injury.

The anterior condyloid foramen is most favorably situated with respect to the course pursued by posterior fossa fractures. Instance of its involvement is extremely rare. Combined injury of the tenth and twelfth nerves have been described with loss of speech and ability to swallow, paralysis of half of the tongue, soft palate and vocal cord, and pharyngeal constriction. In eleventh and twelfth nerve involvement, hypoglossal sternomastoid and trapezius muscle paralysis would be observed.

Fractures of the mastoid process usually show emphysema, pneumatocele, bleeding from or into the middle ear with the escape of blood into the pharynx by way of the eustachian tube. I recall a case where only a marked hemotympanum resulted. Extensive fracture of the mastoid may be associated with injury to the lateral sinus, laceration of the dura and the brain, injury to the aqueductus Falopii and paralysis of the facial nerve. Late effects of fracture of the mastoid may be deafness, suppurative otitis media and chronic mastoiditis with necrosis. In the latter type case, mastoidectomy is indicated. Otherwise reduction of the mastoid fracture is not attempted and surgical intervention in the immediate vicinity of the fracture is indicated by the emergencies which may arise.

FRACTURES OF THE VAULT OF THE SKULL

In many instances vault fractures may be considered as upward extensions of fractures of the base or they may be limited to the vault depending upon the location of the blow, its direction, and its severity. Vault localization usually occurs from a small weapon striking near the vertex with great violence. This explains why compound fractures are more liable to be limited to the vault than simple fractures. Injuries occurring when the head is in motion, as in a fall or a motor accident, give a diffuse injury with at times a remote simple fracture as previously described (contre-coup), the frontal and occipital lobes being most affected. With the head in motion, the greatest degree of contusion to the brain is usually the under surface of the frontal lobes and the tips of the temporal lobes.

Fractures of the vault may involve the external table or the internal table alone, or the whole thickness of the skull.

Fractures of the external table alone are extremely rare giving a gutter-shaped depression. Fractures of the inner table alone have long been recognized and are quite common. Only a moderate force may be necessary by a small body such as a stone. Fractures of the whole thickness of the skull may occur. These may be simple or compound; with or without depression; elevated, fissured, stellate, comminuted, punctured, or explosive.

SYMPTOMS ASSOCIATED WITH FRACTURES OF THE VAULT

A diagnosis of fracture of the vault is made, on evidence obtained by local examination and from symptoms dependent on injury to the intracranial contents. The various intracranial lesions are discussed elsewhere.

Evidence supplied by local examination. A compound fracture will be most readily determined by digital examination, previous to which the scalpwound must be carefully cleansed. Digital examination is greatly preferable to investigation with the aid of the probe. In any case, care must be taken to avoid mistaking one of the sutures of the skull for a fissured fracture.

In simple fractures the diagnosis is frequently obscured by an extensive subaponeurotic or subpericranial hematoma. Irregularities of surface are more or less diagnostic of a solution in the surface of the bone, and a linear hematoma is of corresponding clinical value. In any case, the presence of an extensive hematoma must be regarded as of so suggestive a nature that exploration is called for, more especi-

ally when prolonged concussion or compression is coexistent. Such treatment is imperative when the hematoma—whether diffuse, localized, or linear—pulsates, such a condition implying a breach in the surface of the bone and dura with communication between the extracranial and intracranial hemorrhages.

INTRACRANIAL INJURIES

These comprise extra- and subdural hemorrhages and injury to the brain substance.

Leary[8] has pointed out that the functions of the nervous system require that the system be insulated from other tissues except at the nerve ends and that this insulation requirement as well as the delicate character of brain tissue is probably responsible for the encasement in an essentially closed box. This conception explains much of the symptomatology of intracranial pathology. Since expansion of the skull is not possible, all extraneous or additional substances encroach upon the brain space itself.

In the protection of the brain, the arachnoid plays a unique rôle. It insulates the brain to some extent from the blood by its barrier action; it conducts the arteries over the surface of the brain so that no large pulsating vessels actually penetrate the brain; and it forms a water bath for the brain by containing and distributing the cerebrospinal fluid. Cushing has described this as the third circulation. It serves also in place of a lymphatic drainage mechanism.

In considering the hazards existing within the skull, Leary[8] has pointed out that the larger cerebral vessels are not infrequently faulty in that the vascular media varies in thickness producing weak spots that may undergo stretching with the formation of aneurysms. Aneurysmal dilatation is also favored by the lack of support to the walls on the arachnoid. The dural (meningeal) arteries are set in bony grooves that may be injured in fractures crossing the grooves as already described. The cerebral veins have no unstriped muscle in their walls. They drain into the venous sinuses through bridging veins across the subdural space. Aberrant veins that do not connect directly with the sinuses are often discrete, delicate, threadlike vessels which may rupture from minor injuries in patients suffering from avitaminosis (alcoholics or scorbutics). This is the usual source of subdural hemorrhage. Most interesting is Leary's conception that the skull with its lining dura enters into articulation, not with the bone, but with the brain and its covering arachnoid.

Much of the peculiar symptomatology of intracranial injury is due to the characteristics of the arachnoid. This highly vascular membrane ignores the blood in the subarachnoid space and leaves the job of its removal to the dense avascular dura. If the dura is injured or resected, the area is rapidly repaired without adhesions, provided the arachnoid is not injured. If the arachnoid is damaged, adhesions will follow.

Epidural Hemorrhage. This normally arises from fractures of the temporal convexity of the skull and may be arterial or venous in origin. In the former case, the blood is derived almost exclusively from the middle meningeal artery. When of venous origin, the source is usually a laceration of one of the sinuses of the brain. When blood is poured out from a torn sinus, it tends to occupy, and be widely diffused in, the subdural space. A venous extradural extravasation is of relatively infrequent occurrence, and the pressure exercised on the brain seldom suffices to permit the development of local or general symptoms of cerebral compression. On these grounds extradural hemorrhage may be regarded as almost necessarily of arterial origin and as derived from a torn middle meningeal artery. Rupture of the meningeal artery frees blood under arterial pressure which dissects the dura from the skull as it collects. Thus a time interval elapses between the injury and the progression of symptoms. The history most common is of a fall striking the side of the head followed by temporary unconsciousness. There is then a return to consciousness with headache, sleepiness, stupor, coma, and death unless surgical intervention is employed.

Middle Meningeal Hemorrhage. The middle meningeal artery enters the middle fossa of the skull through the foramen spinosum, and divides after a short intracranial course into two main terminal divisions, anterior and posterior. The anterior branch passes forward toward the anterior inferior angle of the parietal bone, then changes direction and turns upward and backward toward the vertex of the skull. The posterior branch passes horizontally backward—grooving the squamous portion of the temporal bone—toward the posterior inferior angle of the parietal bone.

Throughout their intracranial course the main trunk and its terminal branches are embedded in the outer wall of the dura mater, except in the sphenoparietal region where the anterior branch of the artery usually occupies a channel in the bone between one-half and one inch in extent.

The anterior branch overlies the prerolandic motor area, while the posterior division is related to temporosphenoidal and lower parietal regions—"silent" areas of the brain.

Middle meningeal hemorrhage occurs most commonly in connection with a fissured or comminuted fracture of the temporal region and as the result of direct violence. A fracture, however, is not necessarily present; the hemorrhage may take place on the side opposite to that at which the injury was received (laceration by contre-coup) and both vessels may be involved.

Middle meningeal hemorrhage, uncomplicated by brain injury, is of infrequent occurrence, the associated injuries to the bone and brain confusing the diagnosis. Moreover, the special pressure effects are dependent not only on the presence of serious brain lesion, but also on the nonexistence of a safety valve, such, for instance, as is afforded in a comminuted fracture of the bones entering into the formation of the temporal fossa or of the roof of the middle and external ears. In the former case, blood will force its way into the temporal region, there forming a temporal hematoma that may pulsate while, in the latter case, the blood escapes freely from the external auditory meatus. It should be noted, however, that pressure applied to the temporal hematoma may lead to the development of irritative or paralytic symptoms confined to the muscles of the opposite side of the body, whilst the restriction of aural bleeding by means of plugs inserted into the ear will lead to the early development of compression symptoms.

The amount of blood extravasated varies according to the caliber of the vessel involved and may be from 50 to 100 cc. The clot may be several inches long and an inch thick in extreme cases. In shape the clot is elliptical; in consistency it is either fluid or jelly-like. During the early stages of its formation it can be readily removed. Later on, it adheres to the dura mater and, when removed, leaves the membrane rough and discolored.

Some uncertainty exists as to the relative frequency with which the trunk and the two terminal branches are exposed to injury. There can be no doubt that the typical clinical symptoms in the majority of cases are evidence of injury to the anterior terminal division; this is due to the anatomical relation of the clot to the motor cortex. Injury to the main trunk is a rare occurrence, for the foramen spinosum lies immediately anterior to the petrosphenoidal suture, the course pursued by typical middle fossa fractures.

The attachment of the dura mater to the sides and the base of the skull exercises a most important influence on the direction in which the blood spreads. Firmly adherent to the lesser wing of the sphenoid in front and to the summit of the petrous bone behind, the membrane intervening between these two regions is but loosely attached to, and readily stripped away from, the floor of the middle fossa. As the clot increases in size, it exerts considerable pressure (mechanical) and tends to separate the dura still further from the bone. The anterior and posterior limitations compel the blood to extend first in the outward direction and then upward toward the vertex of the skull. The temporoparietal region is the most frequent site for middle meningeal extravasations. Parieto-occipital and parietofrontal sites are rare.

Middle meningeal hemorrhages yielding typical clinical symptoms are of infrequent occurrence, yet such extravasations are very commonly present in severe lesions of the skull. This is evidenced by the fact that middle meningeal hemorrhage, of a greater or lesser degree, may be found in 20 per cent of all cases of fractured skull, and in 45 per cent of those in which the middle fossa was involved.

Subdural Hemorrhage. This may be diffuse or localized. In diffuse subdural hemorrhage, the blood may be derived from one of the great sinuses of the brain or from superficial cerebral vessels (laceration of the brain). Rupture of a bridging vein causes a slow accumulation of blood in the subdural space. The poor vascularization of the dura is responsible for the characteristic picture which Leary[8] groups in four stages. First, if death occurs within 24 to 48 hours, the blood will be fluid, molding the surface of the hemisphere on which it lies and flattening the other hemisphere against the skull. Second, if the patient lives 1 to 5 days, the blood will have clotted, but is not yet adherent to the dura. Third, if the fatal termination is delayed for 4 to 14 days, the clot will be adherent to the dura and will have undergone a change in color to a copper hue. The fourth stage finds the clot enveloped in a double membrane, one surface attaching it to the dura, the inner surface separating it from the free subdural space and the arachnoid. During the fourth stage the clot may undergo softening. Breaking down of the blood causes an increase in osmotic pressure converting the clot into a cyst under increased pressure.

An important collateral picture results from the poor dural circulation. The inadequate venous drainage causes the backing up of blood in the low-grade granulations, forming giant capillaries which

tend to rupture and produce secondary hemorrhages. These tend to protract the healing process so that a hematoma once established may persist for years. Minor traumatic lesions of the brain may also result in chronic subdural hemorrhages when the blood in the space is not removed before the arachnoid heals by adhesions. With the formation of a closed arachnoid, the mechanism of subdural contusion and laceration hemorrhages corresponds to the arising from rupture of bridging veins. When, however, lacerations of the arachnoid are extensive and the patient lives, the subdural space is cleared of blood within a month by the efficient arachnoid drainage system.

In blast injuries, subdural hematoma and effusion are described by Abbott et al[15] as arising from small bulging veins. Because of the poor absorptive properties of the dura, the hematomas seldom disappear spontaneously. The blood with its higher protein content attracts the cerebrospinal fluid causing a localized increase in fluid volume until a state of Donnan equilibrium is established. The interesting point made by Abbott is that in bomb blast cases, the symptoms of subdural effusion may escape notice and the mental disturbances be ascribed to a functional disorder or a psychoneurosis of war. The patients present a history of exposure to severe concussion, loss of consciousness for a period of a few minutes to several days, persistent headache, memory loss and irritability. There may be a slight facial palsy, passing hemiparesis or a transient change in reflexes. At operation small amounts of old blood and several ounces of xanthochromic fluid are found in contrast to the large amount of blood in subdural hemorrhage.

The superior longitudinal sinus may be torn by the in-driving of the fragments of bone in a comminuted depressed fracture of the vertex, or by wide separation along the line of the sagittal suture. Blood is diffused throughout the subdural space, on one or both sides of the falx cerebri, but always tending to gravitate toward the lower limits of the supratentorial space. The falx and the tentorium will limit the blood to one side of the skull in about 85 per cent of cases.

In the event of the wound being compound, air may enter the sinus. Inasmuch, however, as the sinus pressure is, under normal conditions, positive, this complication is of rare occurrence, unless the patient is in a state of profound shock from loss of blood or suffers from urgent dyspnea.

The lateral sinus may be laid open in any part of its course, more especially at the angle of junction between the lateral and the

sigmoid sinuses in close relation to the occipitomastoid suture, a region not infrequently involved in basic fractures. The blood effused will occupy the supra- or infratentorial space according to the situation of the rent in the sinus wall.

The cavernous sinus is frequently involved in anterior and middle fossa fractures. The blood effused usually escapes into the nose and the mouth. Subarachnoid hemorrhage is not usually due to trauma, but rather to a ruptured aneurysm.

Injury to the Brain. This must always be regarded as by far the most important of the many complications associated with fracture of the skull. A fracture is not necessarily accompanied by brain injury, and laceration of the brain may exist without a fracture. There can, however, be no question that the more severe cases of head injury are almost invariably associated with some degree of brain injury—varying from contusion to extensive laceration, cortical or central.

No part of the brain is exempt from injury but two regions, the antero-inferior aspect of the frontal lobe and the antero-external aspect of the temporosphenoidal lobe as mentioned, show a special liability to contusion and laceration.

The brain may be injured immediately subjacent to the site at which the blow is delivered (direct injury) or at the pole directly opposite that at which the blow was applied (indirect injury).

Direct injury results from the in-driving of comminuted and depressed fragments of bone, from the passage of a foreign body, and from direct transmission and diffusion of forces through the subjacent cerebral substance.

Indirect injury is much more frequent and is difficult to explain. Many theories have been advanced, the most common being that of contre-coup with sudden violent displacement against the opposing bony barrier.

Contusion and laceration tend to be limited usually to the surface of the brain although in severe injury brain disorganization and hematomas may occur in the depths of the white matter.

Penetrating wounds of the skull may result from flying missiles or bayonet stabs. Bullet tracks are usually larger than the caliber of the bullet. Low velocity bullets tend to carry fragments of the inner table of the skull along, whereas high velocity bullets make a cleaner cut path.

Repair of brain tissue does not occur, since it is too highly differentiated. Lacerations of the arachnoid are repaired by adhesions to the dura, forming cyst-like spaces filled with fluid to equalize intracranial pressure. Brain tissue liquifies and as the blood undergoes dissolution, the pigment persists. On removal of the brain, the adhesions of the dura to the arachnoid are broken, revealing tissue losses and yellow walls forming the "plaques jaunes."

LOCALIZING SYMPTOMS OF INJURY TO THE BRAIN

Frontal lobes. In cases where consciousness is retained or regained and the mental faculties not changed by delirium, laceration of the frontal lobe is associated with default in intellectual control and the lesion is usually on the anterior surface. When laceration is confined to the right side, the mental faculties remain unaffected. Laceration of the under surface of the lobe may also affect the olfactory organ. Krieg[7] refers to the frontal lobe as the synthesist of data past and present into the modes of action, attitudes, ideas and thoughts.

The condition of the patient usually prevents one from obtaining early information as to the power of speech. Isolated lesions of the posterior part of the third left frontal convolution result in motor aphasia, a condition characterized by the inability on the part of the patient to speak in spite of the fact that comprehension is unimpaired. The brain injury is, however, seldom so defined as to be characterized by loss of speech without other phenomena. In very close relation to the area responsible for speech is the writing center, situated at the posterior end of the middle frontal gyrus near the center for movements of the hands and fingers. Injury to this center results in agraphia and alexia.

Word-deafness results from lesions implicating the upper temporosphenoidal lobe, and word-blindness from injuries to the supramarginal and angular gyri in the posterior parietotemporal region.

The lesion must be an extensive one if the symptoms included under the term sensory aphasia (word-blindness, word-deafness) are to be evolved. No localized injury to any small area suffices, and the cortex must be deeply involved. Lacerations of the brain are usually of so gross a nature that further differentiation is seldom to be obtained. Here it might be noted that the cortical centers responsible for speech and its accessories, word-seeing, word-writing, and possibly word-hearing, are all situated on the left side in normal right-handed individuals. The corresponding areas on the right side may be regarded as "silent" areas and may be utilized, if desired, for decom-

pression purposes. No operation with that purpose in view should be carried out, however, unless the surgeon is assured that the patient is right-handed in his actions.

Temporosphenoidal lobe. The relation of sensory aphasia to lesions of the temporosphenoidal lobe has already been mentioned. Injury to anterior poles of this portion of the brain, a region frequently involved, may lead to the development of impaired smell and taste, especially if the lesion includes the uncinate lobe and is situated on the left side. In other respects the temporosphenoidal lobe may be regarded as the "silent" area of the brain.

The precentral convolutions. Injury to the precentral or motor area usually leads to the development of definite symptoms: twitchings, convulsions, or paralysis of the face and extremities on the contra-lateral side. In the earlier stages reflexes are abolished. Later on, as the result of degenerative changes in the pyramidal tracts, spasticity, contractures, and rigidity, with increased reflexes, will be observed in the affected limbs. On the other hand, the muscles do not show degeneration. Babinski's sign is generally present.

The postcentral convolutions. These might be expected to lead to various alterations in tactile and muscle sense, in stereognosis, and in the senses of pain and temperature, but the general condition of the patient seldom permits accurate demonstration. Such sensory disturbances are more frequently observed as late results of head injury.

The occipital lobes. Laceration of the occipital lobes may lead to homonomous hemianopia, for which defect the degree of laceration is considerable, involving mainly the medial aspect of the occipital lobe. The greater portion of the occipital lobe may be removed in monkeys without producing loss of vision, and it is only when the lesion involves the parieto-occipital fissure and passes into the occipitotemporal convolutions that loss of vision is permanent.

A slighter degree of occipital injury may lead to subjective symptoms, such as flashes of light or color changes.

The cerebellum. Lesions of the cerebellum rarely permit the development of such localizing symptoms as are observed in cerebellar tumors. Incoordination of movement ataxia, and other symptoms observed in cerebellar tumor formation are, from the general condition of the patient, incapable of demonstration in cerebellar laceration. It is necessary, however, to draw attention to the significance of yawning and gaping. This symptom has been observed in several

recent cases, and, as far as observations go, it is only present in cerebellar lesions.

It is not enough to consider a cranial injury from the angle of the functional anatomy involved, but it must also be considered from the regional physiology. Here the cerebrospinal fluid and the vascular relations may be more important than the anatomic lesion. The fracture itself may be unimportant, but the brain tissue alterations resulting from the vascular changes may be far reaching.

THE CEREBROSPINAL FLUID SYSTEM

Strange as it may seem, the really solid barrier of the brain is the cerebrospinal fluid. An increase in its amount compresses the brain from the outside and the ventricles from the inside, the dura externally and the arachnoid internally. Collateral with this pressure, there is compression of the cortical veins and the dural sinuses with resultant diminished rate of flow and increase in intravenous pressure. Thus the normal status of the cerebrospinal fluid must be rigidly maintained. There must be a continuous balance between the cerebrospinal fluid formed from the choroid plexus in the ventricles and its escape through the foramina of Magendie and Luschka in the roof of the fourth ventricle. . From here the fluid enters the subarachnoid cerebral and spinal spaces not only over the surface of the brain, but also the cisternal dilatations. The subcortical perivascular spaces also supply a small amount of fluid, not cerebrospinal, into the subarachnoid space.

In all, the cerebrospinal fluid normally amounts to about 130 cc. and is produced at a constant rate at positive pressure. Isotonic with blood plasma, it yet differs chemically from it in having markedly less protein, almost no lipoids, ferments or pigments, but many more chlorides. The pressure varies somewhat with the size of the individual. In the normal individual lying on his back in the horizontal plane, it is between 100 and 160 mm. of water or 8-10 mm. of mercury. In the standing position, the intracranial pressure falls to from 50 to 150 mm. of water below the atmospheric pressure while the lumbar pressure is more than double. The zero point is at about the level of the lower cervical vertebrae. The relationship of this hydrostatic pressure to barometric pressure has received too little attention.

Not only is the constant supply of cerebrospinal fluid essential, but it is necessary that there be an uninterrupted escape through the foramina of Magendie and Luschka and its ultimate free absorption

from the subarachnoid space into the large cranial venous sinuses by way of the arachnoidal villi or pacchionian bodies in the cranial vault. These latter are arachnoid branchings into the venous blood stream with perforations so small that the red or white cell will not pass. The positive pressure of the cerebrospinal fluid sends it into the venous blood stream. This pressure must be slightly above intravenous level for the flow to be in the direction of the latter. Thus we have a delicate balance which in the normal individual is rigidly maintained.

Any variation in the factors of this noncompressible system will influence the brain and its coverings; these are as follows: alterations in the volume of fluid, changes in the arachnoid villi, increase or reduction in the available space for the fluid fluctuation of the intravenous pressure, blockade at the foramina of Magendie and Luschka. Most important is the disturbance in rate of absorption caused often by obstruction of the arachnoid villi by red blood cells following hemorrhage into the subarachnoid space. The disturbed outflow of cerebrospinal fluid leads to a rise in intracranial pressure. An abnormal rise in intravenous pressure will produce the same effect. The rise in cerebrospinal fluid pressure then becomes great enough to restore a flow toward the venous blood stream. Encroachment on the size of the space occupied by the cerebrospinal fluid may also produce a rise in pressure. Shrinking of the brain by dehydration will increase the available space and lead to a lowered pressure. The other noncompressible element of the cranial contents, the arterial and venous blood, provides the commonly fluctuating element of the cranial cavity. That the cerebral circulation is not locally controlled but is sensitive to extracerebral factors such as the carotid sinus was not fully appreciated before the important work of Cobb.[4]

Thus the functional anatomy of the skull provides the basis not only for an understanding of craniocerebral injuries, but for cerebral physiopathology as well.

FACTORS IN FRACTURE OF THE FACIAL STRUCTURES

It has already been mentioned that fractures of the anterior cranial fossa frequently involve the frontal or ethmoidal sinus or both and fractures of the middle fossa may traverse the sphenoid. It follows that in all craniocerebral injuries, careful thought should be given to associated involvement of the paranasal sinuses. Conversely, fractures of the facial bones may be associated with intracranial injury, so that it is advisable to consider the possibility of the fracture

extending into the cranial fossae before undertaking immediate extensive repair of the facial injury. Here again the condition of the patient is the first concern and the reduction of the fracture secondary to the intracranial condition.

The convexity of the frontal squama makes depressed fractures the usual occurrence. When the anterior or inferior wall is affected, the deformity may be corrected by wiring and the wound treated with usual surgical cleansing, sulfanilamide ascorbate and closed. The nasofrontal duct usually suffices for drainage and the nose is treated as for acute frontal sinusitis. If drainage becomes inadequate, a small rubber drain can be inserted in the external wound. When the internal wall is fractured, it is treated for the anterior cranial fossa injury as already described. The fracture is exposed and the mucous membrane in the region denuded to avoid the maintenance of a fissure that could carry nasal infection to the meninges. External drainage is essential in addition to chemotherapy. Injury of the ethmoid is frequently associated with fracture of the internal wall and cerebrospinal rhinorrhea should be looked for. The maxillary sinus will be considered in relation to the face.

The anatomic approach to facial injuries gives as interesting data as it has for craniocerebral injuries. The skull may be viewed as a pyramid with the face as the base. In the gorilla, the sharp occipital protuberance forms a real apex. The rim of this facial base presents a series of solid structures, the frontal peak, the outer rim of the orbit, the zygoma and the mandible. Force applied to this circumferential barrier usually drives the fragments inward against a series of weak spots. Thus the frontal fragments go toward the upper orbital rim. The zygoma usually breaks at the thin apex of the body of the maxilla. When the zygoma rotates outward, the transverse diameter of the orbit increases and the lower orbital rim is elevated. If it is rotated inward, the transverse diameter of the orbit is diminished and the orbital rim is lowered producing a very unpleasant looking deformity which, if bilateral, gives a characteristic horse-like appearance to the face and the orbital contents sag. The fractured zygoma may also block the action of the mandible.

The mandible completing the facial circle is next to the nasal bones which lead the list in frequency of injury. This is so despite the fact that it is the strongest bone of the face and next to the petrous bone, it is the densest in the skeleton. The anatomical weak point is the area of the canine fossa where the deep tooth socket and the mental foramen both weaken the body of the mandible. How-

ever, in view of the exposed character of the mandible, it would be more frequently fractured were it not for its circular shape, elasticity, easy mobility and protective inter articular cartilages. The thickened ridges likewise give it support. Fracture of the mandible is usually multiple and may be associated with craniocerebral injury through involvement of the mandibular fossa of the temporal bone (glenoid cavity) direct to the middle cranial fossa or external auditory canal. There may be dislocation of one or both mandibular condyles.

Injuries of the mandible are more frequently complicated by necrosis because of the characteristics of the blood supply. The superficial parts are poorly supplied when compared with other bones. Fractures of the jaw may injure the inferior alveolar artery running in the mandibular canal and associated with the inferior alveolar nerve.

The coronoid and condylar processes are rarely broken because of their protected location. The rami of the mandible also get a degree of coverage from the zygoma while the angle of the mandible and the symphysis are quite heavy.

In facial injuries we have to consider not only the fractures of the bone but also the traction of the masticatory and neck muscles on the bone fragments. These produce characteristic dislocations depending on the site of fracture. Reduction and wiring of the fragments usually give a satisfactory result.

The mandible also protects the maxilla. While fracture of the malar bone is the most frequent, rupture of the alveolar and palatine processes of the maxilla, either single or bilateral, may occur. The corresponding alveolar process is lowered and interferes with the normal bite. This may be connected by wiring with simple or more complicated devices described by Salinger,[12] Kazanjian,[6] Blair,[2] Adams[1] and Straith.[14] Where the zygoma and the apex of the maxilla are involved with lowering of the orbital floor, Shea[13] recommends the elevation of the fragment with a Ritter sound introduced into the antrum through an antrotomy opening in the inferior nasal meatus. After the reduction of the fragment, he maintains the orbital floor in position with an intra-antral balloon inflated with water. The balloon may remain in place for three to five days after which the pressure is released. If hemorrhage recurs or the fragments slip, the balloon may be reinflated after a few hours of rest.

Gill[5] has described a technique of elevation of the fragment by simply grasping it through the skin with a tenaculum. Approaches

through the canine fossa with visible reduction of the fragment, or elevation of the malar fragment from above by passing down from the temporal fossa have been used.

In general, it may be said that if there is no craniocerebral injury, early reduction of the fracture is desirable, but when such complication is present, as little as possible should be done the first week or ten days.

Fracture of the nasal bones and the nasal septum may, in severe cases, also involve the anterior ethmoid. Usually, elevation and reduction of the fragments are sufficient and no further care of the ethmoid is required. Early reduction is desirable. The narrower part of the nasal bone articulates obliquely with the nasal part of the frontal spine and forms a firm heavy base which, together with the frontal process of the maxilla, makes the root of the nose a cranial support. Here it is rarely injured. The lower free edges of the nasal bones are usually thin and at times almost eggshell-like. This is the most frequent site of fracture of the whole skull.

Thus the geometric configuration of the skull, the hydrostatic factors of the circulation, and the developmental anatomy direct the recognition and control of craniocerebral injuries.

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IX

EXTRANASAL BLOCK ANESTHESIA FOR SUBMUCOUS RESECTION OF THE NASAL SEPTUM

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Prompt and thorough local anesthesia for submucous resection of the nasal septum in the majority of cases can be obtained by the time-tested method of applying cotton pledgets of cocaine and adrenalin to the mucous membrane of the nasal septum.

Topical application of cocaine mud to the septal mucous membrane or novocain injections directly into the septum secure the same results. All of these methods of anesthesia are made inside the nasal cavity and have been highly satisfactory in most cases. There are, however, some noses where it is impossible to introduce any anesthetizing agent into the nostril because of a completely blocking anterior deviation of the septum which abuts up against, or is in actual contact with, the lateral wall of the nose. In rare cases the septum may be actually adherent to the lateral wall of the nose because of adhesions, the result of previous ungentle nasal treatments for sinusitis and allied conditions. Into such a nostril it is impossible to introduce a satisfactory local anesthetic. Such septums are often operated on under ether anesthesia because local anesthesia is inadequate and painful. It is for such cases that I propose a prompt acting and thoroughly efficient method of securing local anesthesia by means of an extranasal block injection of the nerves supplying the septal mucous membrane before they enter the nasal cavity, that is, by novocain injections made outside the nasal cavity.

This method for securing local anesthesia is also applicable for high deviations of the septum made by the quadrangular cartilage and the perpendicular plate of the ethmoid bone, for septal spurs where it is impossible to apply topical anesthesia to the posterior surface of the spur, and for horizontally placed vomer ridges where the vomer is almost in contact with the floor of the nose. These devia-

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tions, especially when associated with a lateral twist of the entire nose to one side, do not lend themselves easily to topical applications inside the nasal cavity.

Anesthesia for the septum operation is obtained with this technique by injecting novocain into both anterior ethmoidal nerves at the inner angle of each orbit, and both sphenopalatine ganglia through the mouth by way of the great palatine foramen situated just medial to the upper third molar tooth.

This technique is a new application of an old procedure. It has been used time and time again to secure anesthesia for local ethmoid sinus surgery. If the operator wishes, he may vary the procedure by using block anesthesia on the deviated side of the septum and applying cocaine anesthesia on the concave side of the septum. Or if the obstructed side presents a low deviation, only the sphenopalatine ganglion injection need be made, supplementing this with cocaine cotton pledgets placed high up on the septum inside the nose.

The septum is supplied by the right and the left medial nasal nerves, and the right and the left nasopalatine nerves. The upper anterior part of the septum is supplied by the medial nasal nerve which is a continuation of the anterior ethmoidal nerve. The anterior ethmoidal nerve is a branch of the ophthalmic division of the trigeminal nerve. It passes through the anterior ethmoidal foramen at the inner aspect of the orbit, hugs the undersurface of the roof of the ethmoid sinus through which it passes horizontally and medially, and enters the cranial cavity where it lies embedded in the dura mater on the cribriform plate. It enters the nasal cavity through the nasal fissure at the anterior aspect of the cribriform plate and terminates in a lateral and a medial branch. The latter is the medial nasal branch which supplies the upper anterior part of the nasal septum.

The posterior inferior part of the septum is supplied by the right and left nasopalatine nerves from the right and the left sphenopalatine ganglia. The sphenopalatine ganglion occupies the upper part of the pterygopalatine fossa just behind the posterior tip of the middle turbinate. It is a small ganglion suspended from the maxillary division of the trigeminal nerve. The nasopalatine nerve from the ganglion enters the sphenopalatine foramen, crosses the nasal cavity medially on the antero-inferior surface of the front face of the sphenoid sinus, and then changes its course obliquely downward and forward on the nasal septum, grooving the vomer in its course, to reach the incisor foramen near the front of the hard palate. Here

it anastomoses with the anterior palatine nerve (also a branch of the sphenopalatine ganglion).

If these two nerves on either side of the septum can be blocked before they enter the nose, thorough anesthesia is immediately obtained. The anterior ethmoidal nerve can be blocked at the anterior ethmoidal foramen at the upper inner aspect of the orbit. A 10 cc. syringe mounted with a #25 needle one inch long is introduced through the skin one centimeter above the inner canthus of the eye, just under the bony rim of the orbit, follows the upper edge of the lamina papyracea of the ethmoidal bone, and at a depth of one inch 1 cc. of two per cent novocain is injected between the bone and the periosteum. Proper caution should be taken to draw back on the syringe to be sure that the anterior ethmoidal artery has not been entered. This can be prevented if one injects a short distance above the area of the anterior ethmoidal foramen, that is, above the fronto-ethmoidal suture line. The injection must also not be made outside the periosteum. This can be accomplished by hugging the bone when the needle is introduced.

The nasopalatine nerve can be blocked by injecting the area of the sphenopalatine ganglion through the greater palatine foramen and up the greater palatine canal. The greater palatine foramen is situated just medial to the upper third molar tooth and about one-fourth inch anterior to the posterior edge of the hard palate. The edge of the hard palate can be accurately determined by palpation with the forefinger in the mouth. A 45 degree angle adapter is inserted between the syringe and the needle in order to secure the proper angulation to enter the greater palatine canal. The foramen can actually be localized and palpated as a shallow dimple in many cases by light palpation with the forefinger. It is gratifying to note how often the needle can be introduced into the canal at the first attempt. With a little practice and experience the ganglion is easily anesthetized. One should draw back on the syringe to be sure that a blood vessel in the greater palatine canal has not been entered.

The needle passes up the great palatine canal 1½ inches and 2 cc. of two per cent novocain is injected. The beginner often makes the mistake of introducing the needle too far posteriorly and too near the midline of the palate, the solution thus passing through the soft palate and into the pharynx. If the patient states that the solution can be felt trickling down the throat, the needle should be withdrawn and reintroduced more anteriorly and nearer the third molar tooth. The bony canal can be recognized by the grating sensation

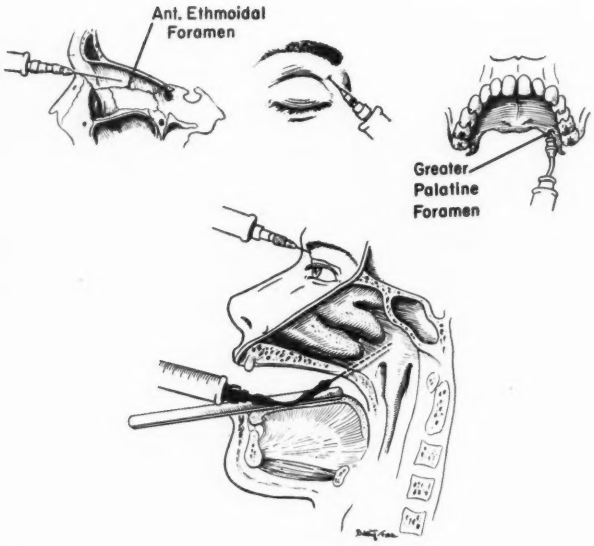

Fig. 1.—Drawings showing: 1) the inner aspect of the left orbit with needle above the fronto-ethmoidal suture line to block the anterior ethmoidal nerve. 2) Anterior ethmoidal nerve injection (front view). The position of the syringe is incorrectly drawn; the syringe should be introduced in a horizontal position. 3) Needle introduced through mouth to block sphenopalatine ganglion. 4) The upper syringe introduced to block the anterior ethmoidal nerve; the lower syringe and the needle introduced through the mouth into the greater palatine foramen to block the sphenopalatine ganglion.

of the needle point as it moves up the canal. The solution should be injected slowly and without undue pressure. No pain is experienced during the injection. No medication whatever need be introduced into the nose to secure anesthesia. However, in order to secure hemostasis, I do inject the septal mucous membrane along the line of the proposed incision with a weak solution (1 to 20,000 dilution) of adrenalin chloride in saline solution. The operation can be started immediately. The anesthesia lasts one hour and is complete.

What are the possible objections to this method of block anesthesia? Some of my confrères have felt that infection in the great palatine canal or thrombosis of, or hemorrhage from, the blood vessels in the canal might follow the introduction of the needle through

an unclean mouth. This has not happened in 200 cases of submucous resection of the septum where I have used it or in numerous local operations on the ethmoid sinus. Neither have I heard of its occurrence from any of my associates. Another possible objection made is that blindness might occur by injecting the optic nerve as it enters the posterior part of the orbit. This can not occur since the needle is only one and a half inches long and can not reach the optic nerve which is situated at least two inches above the greater palatine foramen.

What are the untoward effects which might follow the injection of the anterior ethmoid nerve? Infection of the orbital cavity can be prevented by ordinary cleanliness and has not occurred. If the needle point enters the anterior ethmoidal artery or vein, or if the novocain is introduced outside the orbital periosteum into the orbital contents a sudden tense fullness of the upper eyelid may result. Proptosis of the eye has been observed in a few cases immediately after the injection if the injection is improperly made. This is very alarming to the operator when first seen. However, I can assure you it is only temporary and disappears without any sequelae within a few days. This happened three times in 200 cases. They were among the first patients on which it was tried. It will not occur if the needle is introduced a little higher than the level of the anterior ethmoid foramen (that is, above the fronto-ethmoidal suture line), if the point of the needle hugs the bone on the inner wall of the orbit, and if only a small amount (1 cc.) of solution is injected.

No technique is foolproof. Certain precautions must always be followed in any procedure. Cocaine has been known to cause unpleasant sequelae; even death has been recorded from its use. In my last ten cases the anterior ethmoidal nerve injection has been entirely dispensed with and satisfactory anesthesia has been secured solely with the sphenopalatine ganglion injection. The medial nasal nerve evidently supplies only a very small portion of the septum and probably enters the field of the operation only to a limited extent. However, I am not yet ready to state that complete anesthesia can be secured without the anterior ethmoidal nerve injection.

It must be confessed that hemostasis during the operation is not as thorough with extranasal novocain block anesthesia as when cocaine and adrenalin cotton pledgets are used. However, it is perfectly adequate, and satisfactory visualization of the operative field is obtained.

The needle that is used (stainless steel) can be bent on itself and will not break.

SUMMARY

A technique is described for securing complete anesthesia for the operation of local submucous resection of the nasal septum by block novocain infiltration of the nerves supplying the nasal septum before they enter the nasal cavity. All injections are made outside the nose.

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X

OTOLARYNGOLOGICAL ASPECTS OF BROMIDE AND ACETANILID THERAPY

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Headache is one of the commoner afflictions of the human race; in fact, the commonest cause of patients' seeking medical advice. Some headaches seem to be hereditary, such as migraine, while others are acquired at any age and from multitudinous causes. Of the acquired headaches, some are self-inflicted, whether voluntarily produced or brought on indirectly and innocently by the individual concerned. The greater portion of these latter patients are those individuals who have not sought medical advice and have imprudently attempted self-medication, whether they have formulated a diagnosis of their own or decided to "take a chance" with any one or perhaps many of the numerous headache medications so widely advertised and dispensed at any drug store, soda fountain, cigar stand, ball park, restaurant or hotel—in fact, almost anywhere.

Headaches in children are most likely due to temporary intoxications of varying degree from acute infections, or to diet. The chronic or recurrent headache in a child requires investigation by the ophthalmologist or the dentist, or occasionally the neurosurgeon. Headaches in the adult are varied in cause to such extent that only too frequently the internist, as well as the specialist, has difficulty in locating it, as is well known. There are certain groups of conditions which cause headaches, such as the neuralgias, the vascular conditions, (hypotension or hypertension), the ocular disorders, the intracranial diseases, the intoxications (mainly those of mild obscure chronic infections), the allergic reactions and histamine. Of the intoxications, there are some which are self-inflicted, and these self-inflicted intoxications cause some of the most severe of all headaches. Those individuals present a puzzling situation, and the otolaryngologist, who is quite frequently consulted first, must be well acquainted with this type of intoxication to be prepared to make a diagnosis.

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Headaches are properly and successfully treated according to the cause. The palliative treatment of the acute headache, which occurs during an acute infection, is usually quite sufficient. The medications used are actually very simple, mainly acetylsalicylic acid, alone or in combination with an opiate of choice. These headaches due to acute infections in more recent years have even been found amenable to sulfonamide therapy when such a drug is indicated. The acute headache is also adequately treated with acetanilid or its derivatives. The more chronic and recurrent type of headache due to toxin absorption or to abnormal stimulation of nerves causing neuralgia or neuritis may require more specific medication, particularly the bromides. To cover both the acute and the chronic types of headache, the bromides and acetanilid have been combined in many proprietary preparations. The possibility of habit formation of any drug or intoxication from the same drug, particularly those just mentioned, has been overlooked by individuals prescribing for themselves.

Bromides and acetanilid, or its derivatives, have long been used as medications for the relief of pains and headaches because of their specific action on nerve tissues. The textbooks of pharmacology state that bromides are relatively safe drugs and that a fatality could occur only from an ovedose over a long period of time. These textbooks do admit that toxic symptoms may occur and that the drug is habit forming. The textbooks also describe complications due to intoxication, but skin rash receives more attention and is almost the only complication remembered by a physician from the teachings of his medical school days. The production of headache is mentioned but certainly not stressed.

Acetanilid poisoning is also acknowledged as a definite entity and the same textbooks state that headaches may occur from an overdosage or too often repeated doses. The headache is due to anoxemia and is a similar headache to that which occurs in a profound anemia. The blood becomes cyanotic and it is the cyanosis of the mucosa, which at times not only gives a clue to the diagnosis but also provides for the obtaining of an accurate history, which in itself would be conclusive. Peculiarly, most patients who have not consulted a physician and have taken headache preparations on their own initiative are reluctant to tell the physician the true facts; they admit taking headache preparations containing acetanilid and bromides only after a prolonged and drawn out process of questioning and accusations. The accurate amounts ingested are sometimes never admitted by the patient.

Brominism and acetanilid poisoning have been long and thoroughly described in the medical journals, mainly those journals of the psychiatric or medical services. The otolaryngologist has had to depend on these journals to acquaint himself with these diseases, although he sees more patients suffering with these diseases than any other physician except the psychiatrist. The present-day, war-time, busy physician finds it easier to prescribe the simpler preparations for headaches, and he may be partly to blame for some of the overuse of these drugs by his patients. The average dose, when not too often repeated, of either or both of these drugs, is a safe and rational procedure; but most of the preparations containing these drugs are easily obtained by the patient on refilling the original prescription—and overuse of the drug results. A friend who may have an occasional headache is told about the drug, and this friend purchases the same drug without a prescription and eventually becomes an intoxication sufferer also. There is a tendency to take the headache medications on the slightest suggestion or provocation—or even to prevent a headache.

Many patients having brominism or acetanilid poisoning, or both, complain of sinus infection, and the otolaryngologist in most cases is unable to find such disease. The cyanosis, which is the key to acetanilid poisoning diagnosis, is not easily detected unless daylight, or as near similar illumination as possible, is used. Cyanosis may not be obvious when lipstick is used, and then must be observed in the conjunctiva or the nasal mucosa. Bromide intoxications of mild degree may not be noticeable when there are no marked cerebral changes or skin rash. Therefore, numerous patients complaining of sinus infection are erroneously treated for sinus disease when none is present. A wrong diagnosis also has caused many patients to be fitted with glasses when they were not necessary; likewise, many dental extractions have occurred unnecessarily.

The use of bromide and acetanilid preparations, either singly or combined, is occasioned by a headache or the fear of a headache, but rarely by any other condition. Quite often the individual does not deem a visit to his physician necessary. He is unaware of any sensitivity he may have to either of these drugs. It should be pointed out that individuals vary in their reactions to these drugs and to the efficacy of these drugs as they do to many other drugs commonly used. Some individuals are actually allergic to the drugs. One dose, or perhaps several doses, of the drugs relieves the headache and produces no toxic symptoms. If the preparations are continued, it is probable that toxic symptoms will eventually appear. Headache,

which was relieved by the drug temporarily, again becomes noticeable. The time of reappearance of the headache depends on how much of the drug has been taken and the severity of the intoxication. Hanes and Yates[1] have shown that headache is the most common symptom of bromide intoxication. On account of the recurrent headache the individual takes another dose of the headache preparation and has hopes of relieving all headaches, only to find he has produced another headache—which is a manifestation of brominism or acetanilid poisoning, or a combination of both. Eventually, the individual seeks the advice of a physician; and unless the physician has the history of the drug intake or the complications of skin rash and cyanosis are obvious, it may be very difficult for him to make the correct diagnosis.

The symptoms of bromide intoxication have been thoroughly described by numerous writers, including Craven,[2] Hanes and Yates,[1] and Hanes.[3] Hanes and Yates in an analysis of 400 cases of bromide intoxication in which there was more than 50 mg. of bromides per 100 cc. of blood stated that headache was the chief symptom of which these patients complained. Following in frequency were: irritability, emotional instability, weakness, lethargy, slurred speech, irrelevant speech, delusions, disorientation, hallucinations, loss of memory, cyanosis, vacuous facies, dilated pupils, stupor, blurred vision, fabrication, ataxia, mental confusion, disordered dreams, vertigo, and loss of libido and potentia. It is thus to be noted that headache is practically the only symptom of significance for which a patient suffering with brominism would consult an otolaryngologist. A skin rash was noted in only 25% of the 400 patients just mentioned. It is to be remembered that the normal blood contains only the slightest trace of bromides, if any, and certainly not over 2 mg. per 100 cc.

The extensive use of bromides was shown by Hanes and Yates in studies of blood bromide determinations on 500 medical dispensary patients in Duke Hospital selected at random. There were 64, or 13%, with positive blood bromides, and 29 of these, or 48%, showed 50 mg. or more per 100 cc. They also stated that abnormal blood bromides to the extent of 0.9% were found in the total admissions to Duke Hospital over a period of six and one-half years, there being 700 patients having such abnormal quantities of bromide in the blood. Statistics comparable to these are probably universally found and it is not thought that the above findings are characteristic of any locality.

Blood bromides to the extent of 150 mg. per 100 cc. are said to be very likely to cause psychiatric disturbances for which definite

therapy is necessary. Studies were made of admissions at numerous psychiatric institutions, both state and private, and from 3% to 25% of the total admissions were patients having toxic symptoms of brominism. Many patients suffering with headaches who visit an otolaryngologist are actually ill but are considered neurotics by the examiner. They are mentally disturbed by the bromide intoxication. The diagnosis is brought to light after the correct history has been obtained and the laboratory has reported positive blood bromides and spectroscopic changes.

It is much easier to suspect and even diagnose acetanilid poisoning than brominism because of the cyanosis which appears early in the lips and in the conjunctiva. There is always a history of acetanilid intake or some preparation related to acetanilid, whether the patient admits it or not. The blood spectroscopic examination will show changes, sulfhemoglobin being encountered more frequently than methemoglobin. The diagnosis of brominism is established by the history of intake of preparations containing bromides and a definite diagnosis is made when the blood is found to contain bromides in abnormal quantities.

Individuals vary as to the amount of replacement of chlorides in the blood plasma and the tissues by bromides. The patient who takes only a small amount of bromides may suffer toxic symptoms if he reduces the sodium chloride intake or fails to eat a normal diet. A person imbibing too freely of alcoholic beverages has a tendency to eat less than a normal individual; and if he takes preparations containing bromides for the "hang-over" headaches and has diminished his chloride intake, he is certain to have headaches produced by the replacement of blood chlorides by bromides. Some individuals may take large daily amounts of medicine containing bromides and have little or no toxic symptoms because they also ingest large amounts of sodium chloride.

The treatment of brominism is essentially simple: the taking of large doses of sodium chloride is quite efficient and sufficient. The salt will require the drinking of large amounts of water, which acts as a flushing agent, removing the bromides from the blood. The treatment of acetanilid poisoning is simply the omitting of any further medication containing the drug.

Sodium chloride may be given as plain table salt on the food in large amounts or in capsules and tablets. The latter two methods may be irritating to the stomach unless much water or food is ingested at the same time. Salt is very efficiently given in bouillon.

Since the average adult needs from 8 to 12 grams of sodium chloride daily, Hanes[3] suggests three or four cups of bouillon per day. In the severest cases of bromide intoxication the symptoms should begin to disappear within a week. This applies especially to psychiatric cases where there has been some mental disturbance, because the otolaryngological patient without much psychiatric disturbance would probably be improved in a shorter period of time. Wohl and Robertson[4] have found that injections of desoxycorticosterone, an adrenal cortex compound, will shorten the therapy period.

We have studied the records of 49 patients, all private patients, who were seen on the Otolaryngological Service of Duke Hospital and who had definitely proven brominism and acetanilid poisoning. The disease is found as frequently in the dispensary patient, but only private records were analyzed in this study; they represent consecutive cases. A series of 49 patients is admittedly very small; but, even so, some interesting facts were revealed.

Of the 49 patients there were 47 whose main complaint was that of headache, 3 of whom also complained of nervousness. A generalized headache was the most common type, with occipital and frontal headaches the next most common. Practically all of these patients considered themselves to be sufferers of paranasal sinus infection, and there were 7 patients who had had sinus surgery of some type. There were two additional patients whose antra had been irrigated. There were 6 patients who had definite sphenopalatine ganglion neuralgia, causing unilateral facial pain, which seemed to have been the cause of the initial use of the bromide preparations. In none of these patients were the symptoms relieved for any period of time by the use of bromides and acetanilid, but in practically all cases the medications had been continued in the hopes of obtaining relief. Instead, these patients only increased the severity of the headache already present. There were 8 patients of the group who had definite psychiatric disturbances. There were 2 having questionable disturbance and 1 was extremely worried. The laboratory studies were essentially normal except for the blood bromide and spectroscopic studies. One patient had a hemoglobin content of 62% and another patient had a hemoglobin content of 117%. The hemoglobin content, as well as the blood count, of the remaining patients was within normal range. The blood pressure was found to be normal in most cases, there being one instance of marked hypotension with systolic pressure below 100 and 9 patients with systolic pressure above 150. No patient had any kidney disturbance. Cyanosis of the lips was noted in 35 of the 49 patients. The spectroscopic tests

were not done as routinely as the blood bromides but were proportionately abnormal. Sulfhemoglobin was found more often than methemoglobin. The replacement of chlordies by bromides varied according to the amount of salt consumed by the patient, there being only slight replacement in some individuals who had used large amounts of bromides. The highest blood bromides in the series was 177 mg. per 100 cc. with a replacement of 21. In only one instance was the blood Wassermann, Kahn or Kline reaction positive. The age distribution is interesting in that 13 patients were in their twenties, 17 in their thirties, 12 in their forties, 5 in their fifties, and 2 in their sixties. There were no patients below 20 years of age. There were 24 males and 25 females. There were 8 single individuals and 41 had been married.

SUMMARY

The otolaryngologist is occasionally visited by a patient complaining of a headache for which no otolaryngological cause can be found. Quite often such headaches are produced by intoxications from overuse of acetanilid, or similar preparations, and bromides, or a combination of both. Often these patients have treated themselves with these preparations because they are so easily obtained almost anywhere. The diagnosis is made by the obtaining of a history of such drug intake, cyanosis, and laboratory studies which demonstrate abnormal blood bromides and spectroscopic changes. The treatment requires the discontinuance of these drugs and the taking of large amounts of sodium chloride. It is to be remembered that these drugs are habit forming.

From our study of 49 cases and the studies of the authors quoted, headache is the most common manifestation of intoxication due to brominism and acetanilid poisoning.

DUKE HOSPITAL.

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XI

GENERAL ANESTHESIA FOR TOTAL LARYNGECTOMY

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Several years ago I witnessed two total laryngectomies done under local anesthesia. It seemed that these patients suffered undue pain, psychic shock, as well as a moderate degree of hypoxia. The hypoxia was due to laryngeal obstruction in the early stages of the operation and to collections of blood and to coughing spasms in the later stages of the operation.

Recently I have had occasion to administer anesthesia for four total laryngectomies. With the cooperation of the surgeon a technic was devised whereby the operation was done under general anesthesia, eliminating the undesirable features mentioned above.

Routine pre-anesthetic sedation was employed in all cases where respiratory obstruction was not severe. This consisted of pentobarbital gr. 1½ the night before operation and a moderate dose of morphine and atropine (or scopolamine) 1½ hours pre-operatively. If severe obstruction was present, little or no sedation was used. These patients were anesthetized with nitrous-oxide-oxygen-ether and an ordinary MacGill endotracheal tube inserted. The nasal route is preferred because then it is not necessary to prop the mouth partially open. This allows more room for the surgeon when the head is extended. The gas machine was connected to the endotracheal tube, the head extended, and the operation started (Fig. 1).

The surgeon skeletonized the larynx and upper end of the trachea easily because he could retract these structures from side to side at will with no respiratory obstruction. When this was completed the trachea or lower end of the larynx was opened at the elected site of severance. Under direct vision the anesthetist then withdrew the endotracheal tube sufficiently so that the severances of the trachea could be completed. The cut end of the trachea was grasped with an Allis forceps and brought up into the incision in the neck. The MacGill tube was withdrawn from the larynx. A sterile anode

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This work was done with the cooperation and assistance of the Department of Otolaryngology, Wayne University, College of Medicine, Detroit, Michigan.

tube with a Waters-Guedel type inflatable cuff was then inserted into the exposed end of the trachea by the surgeon. The other end of the tube was handed to the anesthetist. The cuff was inflated and any blood that may have leaked into the trachea was aspirated by means of a well lubricated No. 12 urethral catheter. The tube was then connected to the gas machine and the anesthesia continued in the usual manner with a perfectly clean respiratory tract. At this time it may be necessary to partially redrape the operative field. The anode tube being a latex impregnated wire coil bends freely without kinking so that it was easily pushed out of the way while the operation was completed. (Fig. 2) Excision of the larynx was completed and as the esophagus was being closed a Levine tube was passed through the nose into the stomach. The anode tube was removed for the final step of the operation, which was anchoring the cut end of the trachea.

It was the impression of the surgeon that these patients suffered a minimum of emotional stress. The strain on the myocardium and on the patient in general appeared to be less than when similar operations were done under local anesthesia. Following excision of the larynx, closure of the esophagus and the pharynx is generally conceded to be a tiring and tedious procedure. The patient under local anesthesia is fatigued and may become uncooperative. The efforts of coughing and swallowing are very disturbing to the surgeon. With the type of anesthesia described this important part of the procedure is more readily accomplished by the absence of these disturbing factors.

When partial laryngeal obstruction existed due to carcinoma of the larynx, it did not seem to become worse during induction of general anesthesia. The induction was somewhat slower than usual but when the endotracheal tube was inserted the airway was more patent than before the anesthesia was started. When this operation is done under local anesthesia, the laryngeal obstruction is prone to become worse when the recurrent laryngeal nerves are blocked. We considered, but did not find necessary, the inserting of the MacGill endotracheal tube under topical anesthesia before starting the general anesthesia.

The advantages of this method are: 1) the patient suffers no more discomfort than that associated with induction of a general anesthesia; 2) the psychic effect upon the patient is definitely decreased; 3) the airway is always patent; 4) the surgeon need be hurried at no time; 5) swallowing and coughing are not present to harass the surgeon.

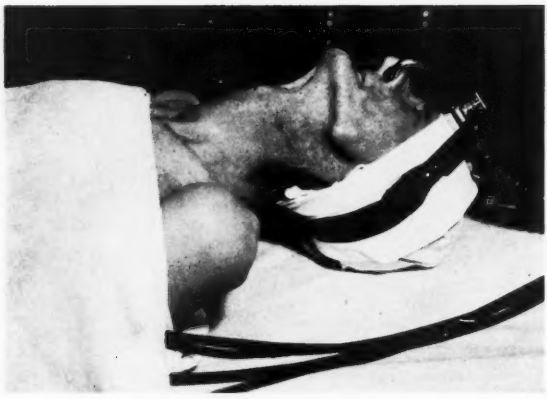

Fig. 1.—The MacGill endotracheal tube through the nose and well down into the trachea is connected to the breathing tubes of the gas machine. The head is extended and the patient ready for surgery. The airway is perfectly free and there is no struggling.

Fig. 2.—The anode tube is shown protruding from the cut end of the trachea and connected to the breathing tubes. Inflation of the Waters-Guedel cuff is maintained by the Bulldog paper clip. The patient's head and the tubes are exposed in this illustration, to show their relations. During the operation only the sterile anode tube is exposed. An unused anode tube and cuff are lying beside the patient.

SUMMARY

General anesthesia for total laryngectomy is described. Endotracheal tubes are employed insuring a patent airway at all times.

RECEIVING HOSPITAL.

XII

THE EFFECTS OF VARIOUS COMBINATIONS OF TEMPERATURE AND HUMIDITY ON ARTIFICIALLY INDUCED PURULENT SINUSITIS IN RABBITS

A LABORATORY EXPERIMENT

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The author, in reviewing the literature concerning the relationships of temperature and humidity to inflammatory diseases of the upper respiratory tract, found a variety of clinical opinions regarding the advantages and disadvantages of various climates and other physical conditions in regard to chronic sinusitis. It was also noted that no objective experimental work concerning the effects of combinations of temperature and humidity on sinusitis had been recorded. These observations stimulated the author's curiosity and he decided to determine, if possible, in an objective manner, the effects of various combinations of temperature and humidity on purulent sinusitis in animals.

Accordingly, an engineering corporation was consulted regarding the preparation and loaning of suitable air-conditioning equipment. Through the kindness of this company, there was designed and constructed a highly satisfactory experimental chamber.

In 1937, under the direction of Dr. L. W. Dean, Professor of Otolaryngology at Washington University, the work was begun at the Buffalo City Hospital, University of Buffalo. Some months later the experimental work was completed by the author under the personal direction of Dr. Dean at the McMillan Institute, Washington University, St. Louis.

The chamber was a suitably insulated box 12′ x 6′ 6″ x 4′, with double refrigerator doors and triple windows. Within it were seven-day constant recording devices for registering dry and wet bulb temperatures. Nearby were rheostats for controlling the desired conditions. The chamber received 18,000 cubic feet of conditioned air

Work done under the auspices of the Department of Otolaryngology, Washington University School of Medicine, St. Louis, Mo.

per hour. This came from a tower with an in-and-out port situated at one end of the chamber. The air within the tower was conditioned by two steam coils for heating and reheating, also by two coils for cooling and recooling. One of the latter coils contained circulating brine and the other was connected with a freon unit. Humidification was accomplished by a fine spray. The various operations of heating and reheating, cooling and recooling and humidification were coordinated electrically and their automatism maintained by the use of solenoid valves. The box had uniform illumination and uniform dust filtration. No attempt was made to maintain a constant barometric pressure.

Rabbits were used throughout this work, and in preliminary experiments with 20 rabbits it was found that a uniform type of purulent sinusitis could be induced as follows: with rabbits under morphine sedation the right antrum was perforated and 1.5 cc. of 1% alizarin red in sterile normal saline was instilled. One-half hour later 1.5 cc. was again instilled. One-half hour later the dye was removed through the ostium by irrigating the antrum with sterile physiological sodium chloride solution until the washing was clear. Then a 1 cc. suspension containing five million each of streptococcus viridans, staphylococcus anhemolyticus and pneumococcus type III was instilled into the sinus. All rabbits were fed the same diet.

Five groups of animals were used in combinations of temperature and humidity which could be classed as: cold and dry, cold and wet, hot and wet, hot and dry, and optimum temperature and humidity. Each group of animals was placed into the desired condition one week after the induction of sinusitis and left there for a period of two weeks, at which time the animals were killed and their heads removed and fixed in 10% formalin. The bony structures were decalcified and frontal paraffin sections through the sinuses made. These were stained with hematoxylin and eosin and were studied microscopically in an effort to evaluate the effect of the atmospheric environment on the sinusitis. In so doing, particular attention was given to loss of cilia, loss of nuclear staining and outline, hydropic degeneration, hyperplasias, thickening of basement membranes, inflammatory cellular infiltration, degree of vascularity, osteoporosis and halisteresis.

RESULTS

Group I. The animals in this group were maintained in an atmosphere of 53° F. dry bulb and 45° F. wet bulb temperature, a relative humidity of 45%, that is, a cold and relatively dry atmo-

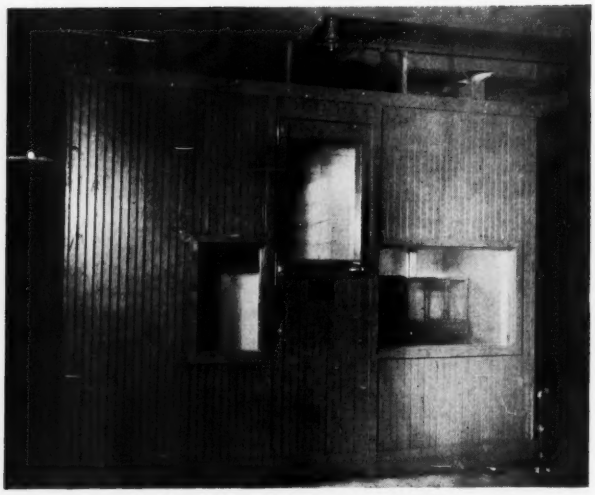

Fig. 1.—Front view of the chamber in which the animals were maintained. Constant recording devices had not been installed at this stage of construction.

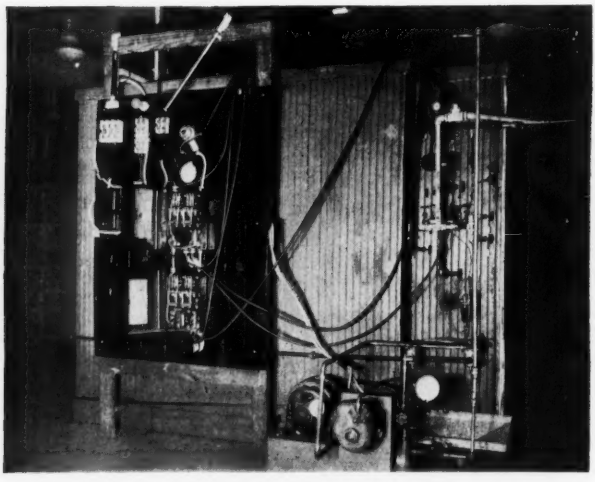

Fig. 2.—Rear view showing the automatic control equipment and the freon unit.

sphere. One of these rabbits died the day after being placed in the air-conditioned chamber. Autopsy disclosed a massive, bilateral empyema. The results of the study of the pathology of this group and the other groups are shown in the accompanying table.

Group II. The animals in Group II were placed in an atmosphere of constantly maintained temperature at 53° F. dry bulb and wet bulb 48° F. The relative humidity was 70%. This could be interpreted as a cold and wet atmospheric environment. One rabbit died three days before the end of the experimental period, was autopsied and found to have an extensive atelectasis.

Group III. The air in the chamber was maintained at 82° F. dry bulb, 75° F. wet bulb temperature, a relative humidity of 73%, corresponding to a warm and relatively wet climate.

Group IV. The fourth group of animals was placed in an atmosphere maintained at 90° F. dry bulb, 60° F. wet bulb temperature, 12% relative humidity, or, a hot and relatively dry atmosphere. Five of these animals died at the end of the third day and one at the end of the fourth day. Autopsy disclosed only generalized parenchymatous degeneration, analogous to that seen in humans in heat exhaustion.

Group V. The fifth group lived in an atmosphere represented as ideal, namely, 70° F. dry bulb, 58.5° F. wet bulb, a relative humidity of 50%. The detailed results of this group like those of the preceding four groups can be noted in the accompanying tables. Incidentally, the author who has a chronic nasal allergy and a low-grade chronic purulent paranasal sinusitis experienced a definitely noticeable degree of lessening of nasal symptoms after entering the chamber to attend Group V.

A histological study was made without reference to the preceding data and the findings tabulated. Scrutiny of the tabulation indicates that of the rabbits in the first four groups, those in Group I, that is, those in a cool and dry atmosphere, presented the least variation and degree thereof in the departure from the structure of the normal maxillary sinus. In one of the rabbits in the first group there was an extensive and severe purulent sinusitis; the exact reasons for this are not clear.

The group of rabbits showing the next greater degree and type of pathologic changes was Group II, or those in a cold and relatively

wet atmosphere (dry bulb 53° F., wet bulb 48° F. and relative humidity 70%). The findings were quite uniform and the degree of pathology was in definite contrast to the third group which was placed in a hot and relatively wet atmosphere.

In this third group which was in an atmosphere of 82° F. dry bulb, 75° F. wet bulb temperature, relative humidity 73%, there was more free exudate and more loss of goblet cells than in the two preceding groups. Thickening of the basement membrane, a sign of tendency to chronicity, was noted. There was, however, less loss of cilia and a greater tendency to hyperplasia of the epithelium.

The fourth group, living in a hot and relatively dry atmosphere (90° F. dry bulb, 60° F. wet bulb temperature and relative humidity of 12%) presented the most extensive and varied degrees of purulent sinusitis. The greatest single histologic constant finding was the leucocytic invasion and dispersion. Loss of cilia and loss of goblet cells closely paralleled this observation. Bone absorption was almost a constant feature.

The fifth group, that is, those in a so-called optimum atmosphere environment, namely, 70° F. dry bulb, 58.5° F. wet bulb, and relative humidity of 50%, presented surprisingly few histopathologic changes.

SUMMARY

Because no objective experimental work regarding the effects of atmospheric environment on purulent sinusitis could be found, some work of this character on rabbits was undertaken. Because of technical difficulties, the effect of all atmospheric factors could not be studied, only those of controlled temperature and humidity were considered.

Histopathologic analyses revealed findings that were surprising. In this study of purulent sinusitis in rabbits it was found that a relatively cool and dry atmosphere was the most beneficial except for the optimum atmospheric conditions set forth in the standards of the American Society of Heating and Ventilating Engineers. A hot and relatively dry atmosphere was the most harmful.

It is emphasized by the author that the above experiment was not performed on large groups of animals. The work was carefully done and clinically evaluated. However, it is felt that the interpretation of the results obtained in rabbits under artificial conditions

cannot be directly applied to humans. It is hoped that this work may stimulate others to proceed with further investigations. In time, a volume of data may be attained which will be large enough to afford more far-reaching conclusions.

The author wishes especially to express gratitude to the late Dr. L. W. Dean, Emeritus Professor of Otolaryngology at Washington University School of Medicine, for his encouragement in this work, and also to Mr. Ralph H. Peo, Vice President, Houde Division of the Houdaille-Hershey Engineering Corporation for the development and loan of the air-conditioning apparatus.

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TABLE 1.—CHANGES SHOWN AT AUTOPSY OF RABBITS

	Rabbit No.	Mucosa a change such as desquamation	Bone absorption	Loss of cilia	Leukocyte invasion	Hyperplasia	Loss of goblet cells	Thickening of basement membrane	Desquamation	Free Exudates
	21									
	22									
	23			+++	+					+++
	24									
	25			S	S					
Group I	26			+	+			+		+++
	27			+	+			+		+++
Cold and Dry	28									
	29									
	30									
	31			+	+			+		+++
	32									
	33		+++	+++	+++	+++	+++	+++	+++	+++
	34									
	35				++					
	36				++					
	37		++		+				++	
Group II	38			S	S	S				
	39			S	S	S				
Cold and Wet	40			S	S	S				
	41			S	S	S				
	42			S	S	S				
	43		+	+	+				S	
	44			+		++				++

TABLE 1— (Continued)

	Rabbit No.	Mucosa a change such as desquamation	Bone absorption	Loss of cilia	Leukocyte invasion	Hyperplasia	Loss of goblet cells	Thickening of basement membrane	Desquamation	Free Exudates
	45			+	++	++				++
	46	+—			S	S				
	47	+—			S	S				
Group III	48		+	+++	+		++++			++++
	49						+			+++
Hot and Dry	50						+			+++
	51				S		S			S
	52			+	+	S	S			S
	53	+—			S	+		S		
	54	+—			S	+		S		
	55	+—			S	+		S		
	56		+	+—	+		+—			
Group IV	57			+	+—		+			
	58		+	+++	+	+	+	+		
Hot and Dry	59				+	+		+		
	60		+	+—	+		+—			
	61			+	+—	+—	+	+		
	62		+	+	++		+	+		
	63		+	+	++		+	+		
	64		+	+	++		+	+		
	65		+	+	++++		+			
	66		+		+	+	+			
	omit									
	68							S		
	69				S			S		

TABLE 1—(Continued)

	Rabbit No.	Mucosa, a change such as desquamation	Bone absorption	Loss of cilia	Leukocyte invasion	Hyperplasia	Loss of goblet cells	Thickening of basement membrane	Desquamation	Free Exudates
Group V	70				S					
	71				S	S				
Optimum	72							S		
	73			S		S	S			
	74			+	+				+	+++
	75			S				S		
	76									
	77									

S, Slight +, Definite +—, Questionable

XIII

GENETIC PRINCIPLES AND THE INHERITANCE OF DEAF-MUTISM

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Washington, D. C.

The growth of our knowledge of human heredity is often retarded by a misunderstanding of the principles of genetics. Such a misunderstanding may involve the misuse of terms employed by geneticists and is likely to result in a failure to interpret properly the pedigrees of families afflicted with hereditary diseases. Erroneous ideas of heredity are thus perpetuated, and doubt may even arise concerning the applicability of the laws of heredity to man.

A proper understanding of the inheritance of congenital defects, diseases and susceptibility to diseases is important to the physician. Accordingly, it seems desirable to discuss the genetic aspects of the pedigree of congenital familial deaf-mutism recently published in this journal, calling attention to misleading statements in the original report[1] and in the discussion published subsequently.[2]

The family under consideration consists of the parents, whose hearing is normal, and nine children; five of the children are designated as deaf-mutes, three have normal hearing, and the status of one, nine months of age, is doubtful. The report states that "There is a negative history of deafness on the side of both the mother and the father," but there is no information as to how many generations the history covers. It was pointed out in the discussion that there is a possibility that some of the children may suffer from deafness resulting from measles, rather than from congenital deafness. If it could be proved that the deafness of all of these children is acquired, a genetic analysis would be superfluous, but so long as any of them suffer from congenital deafness, a genetic explanation is pertinent.

The discussion[2] which followed the presentation of the original report was opened with this statement: "This presentation is a striking example of a type of deafness for which there seems to be no explanation, inasmuch as there is no history of hereditary deafness." In closing the discussion the author of the report said: "This family does not follow the Mendelian law, but there must have been a dom-

inant ancestor that handed down this deafness, probably on the male side; this in spite of the fact that there is absolutely no deafness in the family."

It is the purpose of this paper to point out that there is a simple genetic explanation of this pedigree, based on Mendelian principles. In order to make this explanation intelligible the meaning of such concepts as dominance, recessiveness, homozygotes and heterozygotes must be established. It will then be possible to show how these concepts are utilized in a logical interpretation of the case under examination.

Hereditary characteristics are determined by the operation of one or more hereditary units known as genes. They are located in the chromosomes, and in certain animals which have been carefully investigated the positions of many genes in relation to other genes in the same chromosome have been determined. A fertilized human ovum contains 24 pairs of chromosomes. One chromosome of each pair was present in the unfertilized ovum, the other was brought in by the sperm. The chromosomes composing such a pair are said to be homologous. Genes which are similarly located in each of these chromosomes are called allelic genes; they affect the same parts of the body, but not necessarily in the same way. Let us suppose that one chromosome contains a gene which determines the development of a normal auditory apparatus; if the homologous chromosome contains a similar gene for normal development, then the individual is said to be homozygous for this particular characteristic, that is, he or she is a homozygote with respect to this gene.

But the homologous chromosome may contain an allelic gene for abnormal auditory development (e. g., deaf-mutism) instead. In this case the individual, having a gene for normal auditory development in one chromosome and a gene for abnormal auditory development in the homologous chromosome, is said to be heterozygous or a heterozygote. A further possibility is that both homologous chromosomes may contain a gene for abnormal auditory development. In this case, as in the first one, the individual is homozygous, but with respect to the gene for deaf-mutism rather than with respect to the gene for normal hearing.

Before we can inquire into the effect of such genes on the individual, however, we must understand the meaning of dominance and recessiveness. Except in cases of blended inheritance, which is not involved in this case, only one of the dissimilar genes present in a heterozygous individual is able to produce a distinguishable effect.

This gene is said to be dominant. The other gene, whose effects are held in abeyance, is said to be recessive. Hence it is only in an individual homozygous for such a recessive gene that the effects of this gene become evident.

A brief outline of the growth of our knowledge of the inheritance of deaf-mutism is given by Baur, Fischer and Lenz in their book on human heredity.[3] According to these authors, Albrecht, in 1923, established the fact that deaf-mutism is usually due to a recessive gene, which we may designate by the letter (d). From what has been said in the previous paragraph, it is evident that only individuals homozygous for this gene (dd) will be affected. Individuals homozygous for the corresponding normal gene (DD), as well as heterozygous individuals (Dd), will be normal.

During the maturation divisions which result in the formation of ova and spermia, allelic genes are separated or segregated. The germ cells produced by a homozygous individual are necessarily alike with respect to the gene in question; thus all the germ cells of an individual homozygous for the normal gene will be of one type (D) and those of a deaf-mute will be of one type (d). But heterozygous individuals will produce two types of germ cells (D) and (d) in equal proportions. The expected result of the mating of two such heterozygous individuals is shown in the following diagram:

		Male gametes	
		D	d
Female gametes	D	(DD)	(Dd)
	d	(Dd)	(dd)

From this we can predict that three-fourths of the offspring will be normal and one-fourth will be affected. Furthermore, it becomes clear that if either parent were homozygous for the dominant gene which determines normal hearing (D) all gametes produced by this parent would be alike (all D), all children would receive this gene, and all would be normal, irrespective of whether they received a gene for deaf-mutism from the other parent or not.

We are now in a position to draw some conclusions concerning the genetic make-up of the parents of the children in the particular case under examination. Neither parent can be homozygous for the recessive gene, for if this were so that parent would be affected, and both were said to be normal. Also, neither parent can be homozygous for the dominant gene, since all children would then receive the gene and all would be normal, which is not the case. Therefore, both par-

ents must be heterozygous; they are thus able to transmit the recessive gene to some of their children, but because of the presence of the dominant gene they themselves are normal.

It may be objected that the ratio of normal to affected children displayed by the family described (3:6 or 4:5) does not correspond to the theoretical ratio (3:1) expected in the offspring of heterozygous parents. This in itself does not give sufficient cause for rejecting the explanation just outlined. When we deal with a small number of individuals we should not expect them to show the close correspondence to theoretical expectations which can be obtained with a large number of offspring, as in mass breeding experiments.

On the basis of the fundamental principles of heredity which have been utilized in this discussion, it may therefore be concluded that deaf-mutism in the family under consideration is to be attributed to the operation of a simple recessive gene, as in most other cases of hereditary deaf-mutism. Both parents are heterozygous for this gene, and even though their offspring do not exhibit the theoretical Mendelian ratio there is no reason to assume that Mendelian principles are inadequate to explain the pedigree.

We may now turn our attention to some of the misconceptions concerning basic genetic principles which require special comment. It was stated in the original report that "Heredity is a fifty-fifty proposition in which both partners may be responsible for the future disposition (anlage) of the child. If the determining units or genes, according to Mendel's law, appear equal they are called homozygotic. This means that the father and mother are equally healthy or ill. If these units differ, that is, if the father is ill and the mother is healthy or vice versa, we speak about heterozygotes. If the ill qualities of the child are distinguishable or manifest we call them dominant; if they are latent or not distinguishable we call them recessive."

These statements give an inadequate idea of the meaning of the terms they are intended to explain, largely because they fail to take into account whether the gene responsible is dominant or recessive. Since deaf-mutism is the result of the operation of a recessive gene, an individual will be "ill" only if homozygous for this gene. An individual having only the normal counterpart of this gene would be homozygous but "healthy," while a heterozygous individual would also be "healthy" in spite of the presence of a recessive gene for deafness in his genetic make-up. With regard to the statement concerning dominance and recessiveness, it should be pointed out that though a person homozygous for the gene for deafness will have

"distinguishable ill qualities," it is incorrect to use the term dominant in this connection. Neither should the term recessive be applied to an individual in which "ill qualities" are not distinguishable, for such a person may be either homozygous for the normal gene or heterozygous. Dominance (or recessiveness) is a permanent attribute of the gene, which determines the mode of inheritance. The term dominant should not be applied to individuals, as did the author of the report when he stated in the discussion that "there must have been a dominant ancestor that handed down this deafness."

One of the features of this pedigree which contributed to the confusion concerning its interpretation is the absence of a history of hereditary deafness. From what has been said it should now be clear that this is not to be considered unusual in families exhibiting hereditary diseases dependent on recessive genes. If this pedigree were more extensive we might expect to find in it deaf-mute ancestors of both parents, but since the gene may be passed down through a long line of heterozygous normal individuals the existence of such ancestors may be unknown to the living members of the family. It should be pointed out, however, that there is no justification for assuming that such a deaf-mute ancestor (but not "a dominant ancestor") was "probably on the male side."

In this connection, a few comments on the role of consanguinity are pertinent. It was correctly stated in the discussion that consanguinity of parents "cannot be considered an absolute cause of deafness in the children," and that "the children of consanguine parents are not always deaf if the parents are normal and healthy individuals." From these statements it might be inferred that in certain instances consanguinity alone may be a cause of deafness, which is not true. We can understand why this is not true if we consider that parents both of whom are homozygous for the normal allelic gene (DD) cannot have affected children (dd) no matter how closely they are related, while heterozygous parents (Dd) are likely to have deaf-mute children whether they are related or not. In other words, the characteristics of the children are dependent upon their genetic constitutions, which are dependent in turn upon those of their parents; relationship is incidental.

The high proportion of deaf-mute children from consanguineous marriages is significant because it is a further indication that this characteristic is a recessive. Statistics show that recessive characteristics appear more frequently in the offspring of such marriages than in the general population, while the incidence of dominant character-

istics is not increased. This is accounted for by the circumstance that in order for a child to be affected, both parents must possess the recessive gene; if they are related there is a greater likelihood that they will have this gene than there would be if they were unrelated.

It is hoped that this discussion is sufficiently clear to demonstrate the adequacy of recognized genetic principles for the explanation of such cases. Further information concerning the fundamentals of genetics may be obtained from an article by Macklin,[4] who also discusses common errors and misapprehensions. The extensive treatise on human heredity by Baur, Fischer and Lenz[3] contains a section dealing with other diseases of the ear in which there is evidence that heredity also plays a part.

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XIV

ELIMINATION OF INTRANASAL PACK BY THE TOPICAL USE OF THROMBIN

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The use of thrombin as an aid in operative surgery has been recorded by Tidrick[1] and also by Putnam.[2] Using this purified clotting agent in general surgery Tidrick found it of value in controlling bleeding surfaces especially in skin grafting and in work where cancellous bone was involved in the operative field. Putnam used thrombin in brain surgery where it was combined with an absorbable cellulose fiber and in this combination was found to be of considerable value in the control of bleeding during the operation. The present report deals with the influence of thrombin not only during the operation but also upon the postoperative course of patients following intranasal surgery for drainage of the accessory nasal sinuses.

Recently Crowe[3] has pointed out that the operated areas are less subject to the formation of polyps, adhesions and heavy scars when tyrothricin and penicillin are used to control postoperative infection. His technique employed packing soaked in tyrothricin solution and then irrigations with tyrothricin or penicillin during the healing period.

This postoperative routine was adopted by the author and packing soaked in tyrothricin solution was employed following sinus drainage operations in a series of 19 cases. It proved the value of this agent in the control of infection and in the healing process. Irrigation of the operative field with tyrothricin solution following the removal of the packing also favored healing. It was observed, however, that tyrothricin had a tendency to increase the amount of postoperative bleeding in these cases. While it was necessary to repack the sinuses in only two cases out of the series, there was a definite impression of more oozing when tyrothricin-soaked packing was applied.

A supply of thrombin came to hand about this time and seemed to offer an agent of value following sinus drainage operations. This thrombin had been produced from bovine plasma by the method of Seegers[4, 5] and was obtained as a dry powder which when dissolved

had considerable clotting power. In the present series 10,000 Iowa units of this powder was dissolved in 10 cc. of saline and this solution was then sprayed onto the operative field.

When thrombin was used during operations it was found to give a firm clot and to control bleeding well but when suction was used to clear the operative field for visibility the bleeding started again. Little, if any, gain was obtained from this manner of use and there was a loss of time thereby. Thrombin was, therefore, not of sufficient aid to adopt as a routine measure during an operation.

Thrombin was then used with the idea of controlling the post-operative oozing associated with the tyrothricin packing. It was applied in the following manner. At the termination of the operation the field was well covered with tampons soaked in ephedrine-adrenalin* solution to bring about vasoconstriction. After the removal of these tampons the field was thoroughly suctioned and the thrombin solution was then sprayed freely over the area. After a delay of about three minutes the packing soaked in tyrothricin solution was introduced into the nasal chamber. The results were disappointing where thrombin and tyrothricin were used. In the five cases tried, the oozing seemed to go on much as it did without the use of thrombin. There was no striking lessening of the bleeding when this agent was used. This was attributed to the tissue surfaces covered by the clot being disturbed by the packing's being introduced over them and also by the adverse influence of the tyrothricin upon the clotting mechanism.

The use of thrombin combined with tyrothricin packing was then abandoned and the following technique was followed upon a suitable subject. After the operative work was completed (submucous resection of the septum and intranasal drainage of the maxillary sinuses, local anesthesia) tampons freely soaked in ephedrine-adrenalin solution were inserted into the operative field and allowed to remain for a short time. The tampons were then removed and suction was applied to make the field as dry as possible. Thrombin was then sprayed thoroughly over the operative field in the proportion of 10,000 Iowa units to 10 cc. of saline solution. After the oozing was controlled in this way the external nares were closed with plugs covered with petrolatum to prevent the passage of air and so insure against disturbance of the operative field. Twenty-four hours later the external nares plugs were removed and the nasal chamber and exposed sinuses irrigated with 1/20,000 tyrothricin solution. Similar irrigations were done daily.

*Adrenalin 1 cc.; ephedrine sulfate, 3% solution, 4 cc.; normal saline 25 cc.

The results obtained from this technique were quite satisfactory. It was found that the postoperative bleeding was less than is usually seen in this type of operation. The patient was able to breathe moderately through each side of the nose as soon as the external nares plugs were removed and he never entirely lost this airway. The postoperative swelling of the nasal membranes was decidedly less than usually seen. The patient was more comfortable. The healing and the return of the nasal chamber to its normal function took place in a much shorter period of time.

The advantages of this modification of technique based upon the use of thrombin seemed so definite that it was employed upon a series of cases which now totals 27.

From a study of these cases the beneficial results obtained on the first case in which this technique was employed were repeatedly observed. In each instance there was generally less bleeding after the operation than when packing was employed. The patient had greater comfort by far. There was less injury to the nasal tissues as indicated by the diminished swelling and more rapid healing. A useful airway prevailed after the external nares plugs were removed almost without exception.

Another advantage of this postoperative technique with thrombin was the relief from pain and additional bleeding associated with the removal of the packing. General anesthesia (nitrous oxide or vinethine) was often used to relieve this pain during the removal. This, however, was another set-back to the patient within 24 to 48 hours after the original operation. The elimination of this entire procedure was accomplished when thrombin was used and the need for packing thereby removed.

From these observations it appears: that the use of packing in intranasal surgery led to interference with the function of, and probably caused injury to, the nasal membranes. (This occurs principally by interfering with circulation in the packed area.); that this packing retarded the return to normal function of these membranes and prolonged the period of discomfort and healing; that thrombin gave a clot of sufficient tenacity to make the use of intranasal packing unnecessary in operative cases; that the use of tyrothricin solution as a daily irrigation during the postoperative period was beneficial; that healing was more rapid and the patients had greater comfort when thrombin was used in the manner outlined.

421 HUGUENOT STREET.

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XV

A HOMEMADE CAMERA FOR KODACHROME LARYNGEAL PHOTOGRAPHY

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ST. LOUIS, MO.

Following is a description of a serviceable laryngeal camera built at a cost of twenty-five dollars.

In order to obtain Kodachrome transparencies of laryngeal disease, an instrument was constructed as outlined by Brubaker and Holinger[1] using only scrap materials and household tools in order to keep the total cost within a house-officer's means.

A camera designed for this purpose should include: (a) a viewing system; (b) a photographing system; (c) a source of viewing illumination; (d) a source of photographing illumination. Both sources of illumination should be co-axial, i.e. directed along the viewing axis.

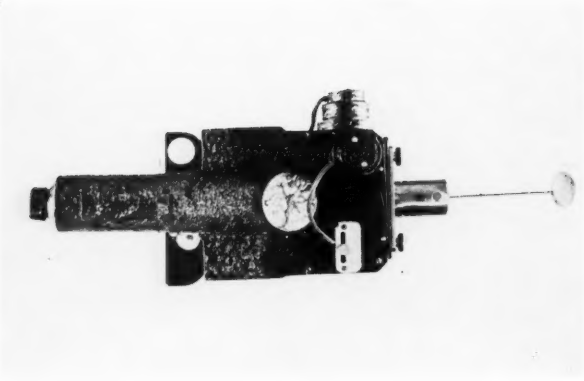
A preliminary model was assembled from cardboard to obtain the proper dimensions, using a ten-dollar, 3½ inch Wollensack lens and a 35 mm. film-carrying-back from an inexpensive popular camera.

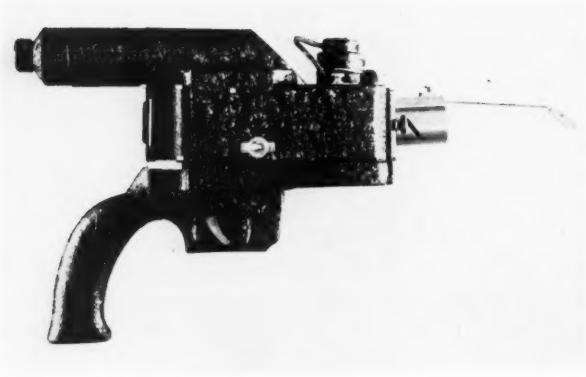
The final model was constructed using a ¾-inch oak board for a base, a ⅛-inch steel angle as a support for the laryngoscope or mirror, and a wooden saw handle to serve as a grip. The case was fashioned from appropriately cut and soldered tin cans, and finished in black wrinkle finish enamel.

Viewing System.—This is a telescope raised above the axis of the instrument by two 45-degree mirrors and employs the camera lens and a plus 16 spectacle lens which, of course, results in an inverted image.

Photographing System.—By raising the lower 45-degree mirror by means of a trigger the viewing system is closed and the photographic system opened from the camera lens through to the 35 mm. film.

From the department of Otolaryngology, Washington University Medical School, Saint Louis, Missouri.





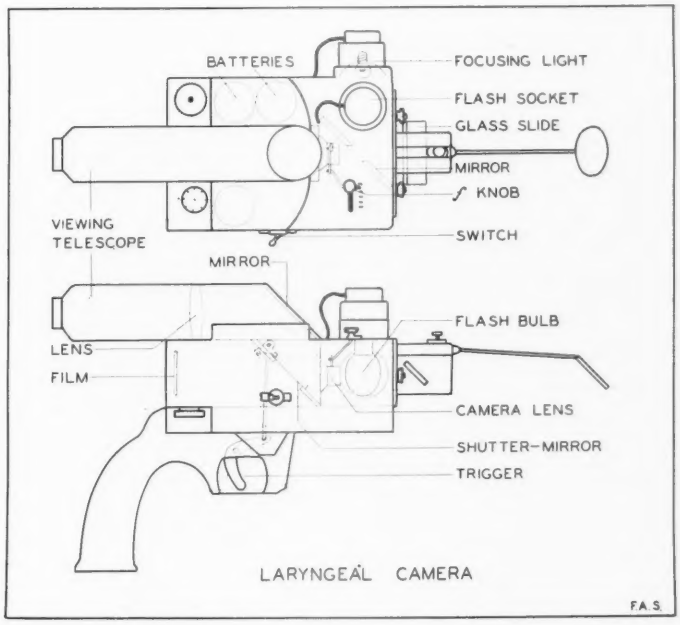


Fig. 3.

Viewing Illumination.—When the movable mirror is in the viewing position, contact is made through a three cell flash-light which serves as a source of viewing illumination. This light is directed axially by a third 45-degree mirror (horizontal plane) in front of the camera lens. A small opening in this mirror permits both viewing and photographing.

Photographing Illumination.—When the movable mirror is raised by the trigger, the viewing light is extinguished and the exposure is made automatically with a No. 5 midget photoflash bulb. This light is also directed axially by the perforated mirror.

Releasing the trigger re-establishes the viewing illumination.

The focus is fixed at 9 inches and the exposure is the full length of the flash or about 1/50 of a second.

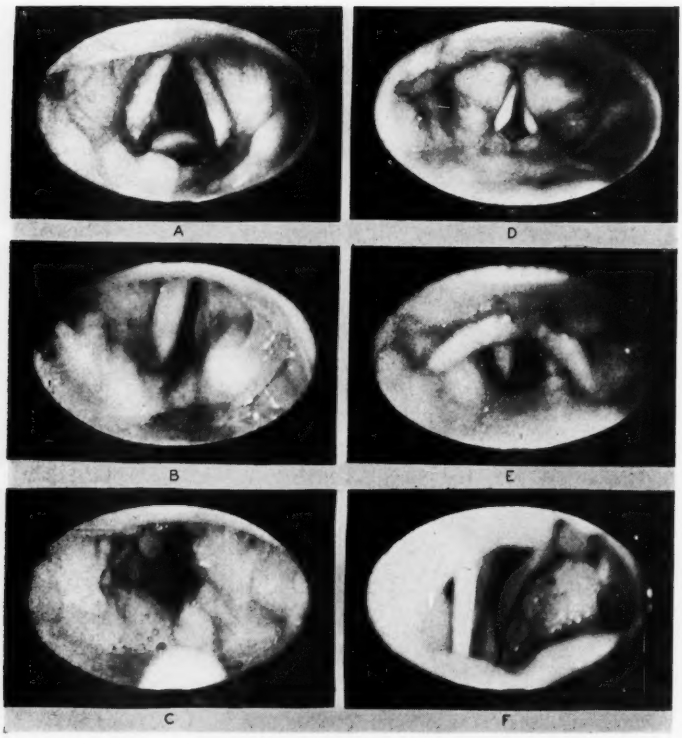


Fig. 4.—Black and white prints made from original Kodachrome transparencies. *A* Normal male larynx. *B* Paralysis of the left vocal cord due to a "party wall" carcinoma. *C* Far advanced laryngeal tuberculosis with extensive ulceration and complete slough of the epiglottis. *D* Normal female larynx. *E* Postoperative view of a female larynx after a left King laryngeal plastic operation for bilateral laryngeal paralysis following thyroidectomy. (Color difference in the abducted cord is due to edema). *F* Nasopharynx, "mulberry hypertrophy" of inferior turbinate.

The light is regulated by an externally controlled iris diaphragm. An aperture of f/16 was found satisfactory for most indirect pictures in adults, and admitted enough light for viewing purposes.

A removable glass slide as employed by Brubaker and Holinger prevented fogging, and this was placed at a 45-degree angle in front of the lens to reduce flare.

The flashbulb color temperature of about 3800 Kelvin was reduced nearer to the 3400 Kelvin needed for type A Kodachrome film by means of an Omega Chrome flash filter attached between the camera lens and the shutter. The resultant color rendition is reasonably accurate.

TECHNIQUE

The patient is placed in position and asked to hold his own tongue. The glass slide and mirror are heated and the mirror with the camera attached is introduced under direct vision through the telescope. When the field is properly centered it is photographed by pulling the trigger.

Topical anesthesia to the pharynx is used in the presence of an active gag reflex.

A direct laryngoscopic attachment was made but has been discarded in favor of the indirect method for convenience.

Inclusion of batteries in the instrument adds slightly to the total weight of five pounds but is much more convenient and eliminates the risk of using house current through a rheostat or a transformer.

The trigger action is for convenience and to eliminate the need of an assistant.

SUMMARY

A camera for laryngeal photography in color similar to that described by Brubaker and Holinger[1] is presented.

Chief emphasis has been placed on simplicity and low cost.

The photographs obtained leave room for improvement but have proven of value for teaching purposes.

640 South Kingshighway.

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Clinical Notes

XVI

LARYNGOCELE—ASSOCIATED WITH CANCER OF THE LARYNX

Case Report

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Since Larrey,[1] in 1829, first described laryngeal air sacs in man, the subject of aerocele or laryngocele has been well presented by a few authors[2-4] and case reports have been given by others.[5-9] The anatomy, theories as to etiology, classification, symptoms and treatment have already been discussed. The comparative anatomy of laryngeal air sacs both in animals and in man has been thoroughly studied by Negus.[10]

The condition, however, is not uncommon. Authors[12] writing on the subject of laryngocele in 1923 state that 35 cases had been recorded. Lothrop,[7] in 1943, reports a total of 80 cases appearing in the literature.

Although the condition is not uncommon, there are few anatomical specimens. Watkins[2] in his review on "Laryngocele in Man" states, "Considering the size of the literature on the subject it is surprising that more information is not available on the detailed anatomy of these cysts. Few details of the anatomy are available and only one museum specimen that might be a laryngocele has been found. The possible specimen . . . is in the Royal College of Surgeons of England and is described as a cyst of the larynx." Wheeler,[5] however, publishes a drawing of a superior laryngocele from a museum specimen in Trinity College, Dublin.

In 1939 two cases of laryngocele, one unilateral, the other bilateral, were seen at the Massachusetts Eye and Ear Infirmary. These two cases have been reported by Allman and Cordray.[6]

Read before the Eastern Section Meeting of the American Laryngological, Rhinological and Otological Society, New York City, January 14, 1944.

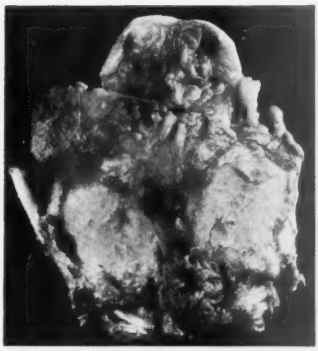

Fig. 1.—Anterior-posterior x-ray view of neck, showing a right superior external laryngocele.

Fig. 2.—Lateral x-ray view of the neck, showing a laryngocele.

This paper is a report of a third case seen at the Infirmary and from a careful review of the literature it is perhaps the first report of a laryngocele found in association with cancer of the larynx.

REPORT OF A CASE

A white male, aged 61, gave a history of having been hoarse for over two years. Six months before entry he underwent a direct laryngoscopy and a new growth was found involving the entire right cord, with fixation of the cord. A biopsy was done and the tissue was reported to be epidermoid carcinoma grade II. There were no palpable glands. A total laryngectomy was advised and refused.

The partient came to New England and six months later, because of respiratory difficulty, consulted Dr. Harold G. Tobey. When seen by Dr. Tobey he was in respiratory distress but, due to an extensive edema of the arytenoids and a smooth mass on the right side of the larynx, a view of the cords could not be obtained. An x-ray examination of the neck showed not only an obstructive lesion of the larynx, but a right superior external laryngocele as well. A tracheotomy was performed. In doing the tracheotomy, a small innocent appearing gland was found and removed. Microscopically the gland showed metastatic cancer. While recuperating from his

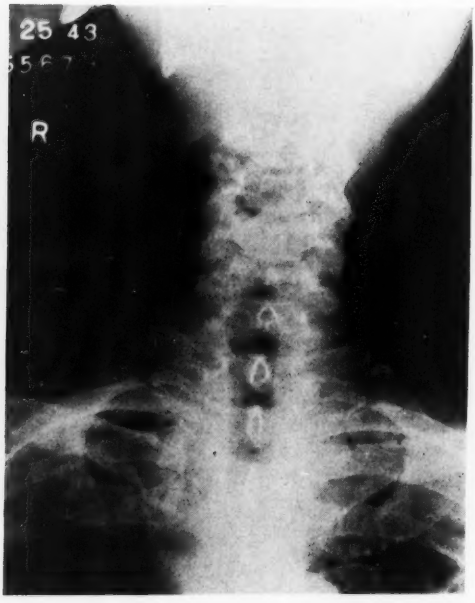

Fig. 3.—An anterior view of the larynx, showing a right superior external laryngocele, which perforates the thyro-hyoid membrane.

tracheotomy the patient developed a sensation of a lump in the throat when he swallowed and a soft cystic mass could be palpated on the right side of the neck just above the thyroid cartilage.

The patient was seen in consultation and a total laryngectomy was advised.

On October 26, 1943, at the Massachusetts Eye and Ear Infirmary a total laryngectomy was performed under avertin anesthesia, supplemented with ether. The larynx was exposed with the adult Jackson laryngoscope and the cords were found to be obscured by very edematous arytenoids and a smooth mass filling the right side of the larynx above the true vocal cord. The right cord was fixed and the entire cord was involved by a proliferating new growth which spread up over the anterior commissure and over the left false cord. A No. 3 Jackson bronchoscope was passed between the cords, the tracheotomy tube was removed, and the bronchoscope passed almost to the carina. Through a T incision the larynx was removed

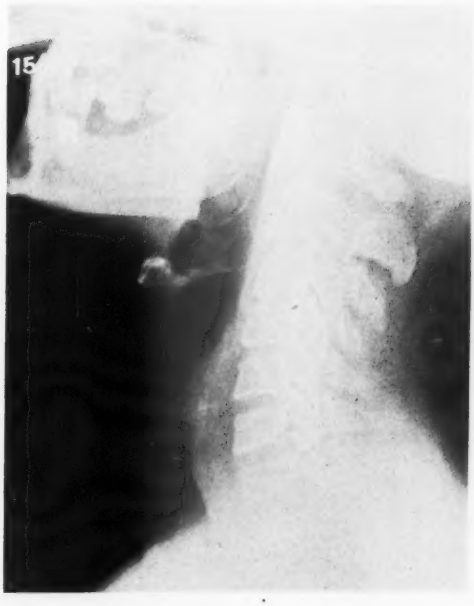

Fig. 4.—A view of the laryngeal lumen, showing extensive carcinoma and a right superior external laryngocele.

including the pretracheal muscles, the hyoid bone, and the epiglottis. The cervical glands along the carotid sheaths were not palpable. The usual closure procedure, with sulfanilamide powder in the wound and lateral drainage, was carried out. Healing was by first intention, and the patient was discharged in three weeks. The X-ray Department advised against postoperative irradiation, unless recurrences develop.

PATHOLOGY

The larynx, the epiglottis, and a right superior laryngocele were removed en masse. The carcinoma involved the right vocal cord, the subglottic space for a distance of 0.5 cm., the right ventricle, the anterior commissure, spread up to the tubercle of the epiglottis and extended over the anterior half of the left false cord. The laryngocele was of the superior type, perforating the thyroid-hyoid membrane. In the fresh specimen the external sac measured 3 x 2 cm. A probe could be passed from the sinus of Morgagni to the dome of

the sac. Microscopic examination showed it to be lined with respiratory epithelium—hence a true laryngocele.

DISCUSSION

Laryngocele in association with a polyp of the sinus of Morgagni has been described.[11] Other cases have been associated with cough,[12, 16] with glass blowing,[13] with lifting weights,[14] and with the playing of wind instruments.[15] Increased intratracheal pressure, as demonstrated by Lindsay,[4] may be a factor in the production of these sacs. Perhaps the obstruction produced by the new growth in this patient may have been a factor in producing an increased intralaryngeal pressure that influenced the development of the laryngocele.

However, as Watkins[2] states, "The resemblance between superior external laryngocele and the laryngeal sacs of anthropoid apes is so great that one cannot but believe that there is a connection between the two."

The reports of laryngoceles occurring in young children, of from one to thirteen years of age,[9, 12] give support to the theory of congenital origin.

CONCLUSION

A case of an external superior laryngocele in association with cancer of the larynx in a 61-year-old white male is recorded.

243 CHARLES STREET.

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XVII

SYNDROME OF AVELLIS

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BOSTON, MASS.

Diseases of nerve centers have been described as combinations of paralytic phenomena, each including involvement of the recurrent laryngeal nerve. They are of interest only because of combinations of paralyses. It has often been stated that there are but few laryngologists and neurologists who, without consulting books, can tell exactly the difference between the syndromes of Jackson, Collet, Tapia, Avellis, Vernet and Schmidt. Even those who write about them often confuse them. These names are impractical, incorrect and misleading. They are merely of academic interest. When, by a progressive process at the cranial base, today the vagus, tomorrow the accessory, later on perhaps the hypoglossal and eventually the glosso-pharyngeal nerves are attacked, it is aimless to speak of a transition of the syndrome of Avellis into that of Schmidt, into that of Jackson, or into that of Collet. As each of these can be accompanied by disorders of the sympathetic, facial and auditory nerves, not excepting those of the pyramidal tract, the list remains hopelessly incomplete. Such progress may be observed in degenerative diseases of the medulla oblongata as with syphilitic and neoplastic processes at the cranial base, or even in disturbances outside of the cranial vault.

Reference to the charts, prepared by Burger, shows the neurologic combinations for the various syndromes. (Charts 1 and 2.)

The appearance of laryngeal paralyses often enables one to foretell that some disease of the brain is on the way long before the disease manifests itself, because recurrent nerve paralyses may be associated with affections of the facial, hypoglossal and oculomotor nerve roots. In diseases of the nerve centers (central) the paralyses are usually unilateral, but not infrequently bilateral. Bulbar lesions usually result in bilateral paralyses. In bulbar paralyses if the tongue is paralyzed, the deviation is homolateral. Unilateral paralysis of the soft palate results in its being pulled upwards and sidewards to the unaffected side.

Read before the 38th Annual Meeting of the New England Oto-Laryngological Society, February 10, 1943.

NEUROLOGIC (ASSOCIATED VOCAL CORD PARALYSES) COMBINATIONS. SYNDROMES OF ASSOCIATED PARALYSES OF THE LARYNX (AFTER BURGER).

SYNDROME	PARALYSES OF NERVES				
	IX	X		XI	XII
	TASTE	VOCAL CORD	PALATE	SHOULDER	TONGUE
Collet & Sicard	+	+	+	+	+
Hughlings & Jackson		+	+	+	+
Vernet	+	+	+	+	
Schmidt		+	+	+	
Avellis		+	+		
Tapia		+			+
Villaret	+	+	+		+

CHART I

ANATOMIC (ASSOCIATED VOCAL CORD PARALYSES) CONSIDERATIONS. ANATOMIC SITES FOR LESIONS WHICH CAUSE THE VARIOUS ASSOCIATED PARALYSES (AFTER BURGER).

	SYNDROME	PARALYSES IN DOMAIN OF NERVES	VAGUS LESION
A	Bulbar	IX, X, XI, XII (V, VI, VII, VIII)	High vagus paralysis
B	Jugular foramen	IX, X, XI, XII Sympathetic	High vagus paralysis
C	Parapharyngeal space	IX, X, XII, (VII) Sympathetic	Deep vagus paralysis
D	Arch of aorta	X, Phrenic, sympathetic	Recurrent laryngeal nerve or deep vagus paralysis

CHART II

The diagnosis of the syndrome of Avellis is based on the following clinical manifestations:

1. Paralysis of the soft palate, partial paralysis of the constrictors of the pharynx, paralysis of the vocal cord and partial paralysis of the esophagus. These paralyses are ipsilateral to the lesion.

2. Contralateral loss of pain and temperature sensibility of half of the body below the interauricular line.

3. The retention of all types of somatic sensation in the areas showing defects in pain and temperature sensibility.

4. Chronic endarteritis.

5. The absence of all other symptoms, that is, motor and sensory disturbances.

There is not a tremendous amount written about Avellis's syndrome. Lederer[1] of the University of Illinois mentions it in his book. Imperatori[2] described three cases before the American Laryngological Association Meeting held in Swampscott, Massachusetts, in 1924. He gave the case histories of three patients from three different hospitals.

REPORT OF A CASE

On the morning of January 9, 1943, when in the Out-Patient Department of the Massachusetts Eye and Ear Infirmary, I picked up a letter from the patient's doctor, Dr. John J. Hilton of Lawrence, Massachusetts, who briefly stated, "For the past two weeks the patient has had difficulty in swallowing and has been hoarse." That sentence put me on the alert, because the two symptoms were present at the same time.

The patient is a 48-year-old white female, married, who pairs or matches stockings in a nearby mill. The family history is negative and she had always enjoyed good health until December 23, 1942. On that evening, while eating steak for supper, she noticed an inability to swallow any solids and that fluids passed down only very slowly. There was no history of any foreign body. On December 25, just two days later, she noticed a change taking place in her voice, but at no time was there a true aphonia. There has never been a return to her true voice tone. The dysphagia has remained a constant symptom. Attempts at swallowing fluids did not produce any regurgitation as the patient quickly learned to swallow only very small amounts at a time.

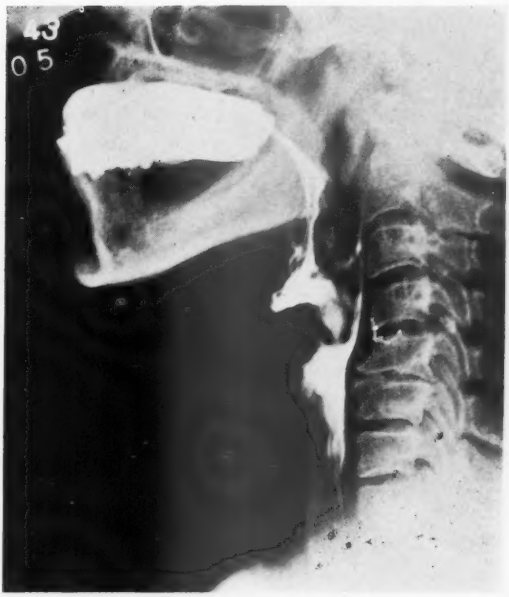

Fig. 1.—Roentgenogram, lateral view, showing collection of barium in the vallecula and the pyriform sinuses. The upper end of the esophagus shows normal markings.

Upon examination, the nose was essentially negative and the soft palate on the left side was not sensitive when touched by a tongue depressor. The tongue movement was questionable. The gag reflex did not seem present and the tonsils were apparent. The laryngeal examination was very easily accomplished and revealed a swelling about the left arytenoid. There was no movement of the left vocal cord. The right cord appeared to be functioning but seemed to stop abruptly in the midline. The interarytenoid space did not seem to be involved. The presence of mucus was rather marked, and the patient had to "clear her throat" frequently. There had been no pain at any time. The blood pressure was systolic 180 mm. of mercury and diastolic 120 mm. The routine blood examination was negative. A fluoroscopy was done by Dr. Alexander S. MacMillan and a paralysis of the muscles of deglutition was amply demonstrated. (Figs. 1 and 2.)

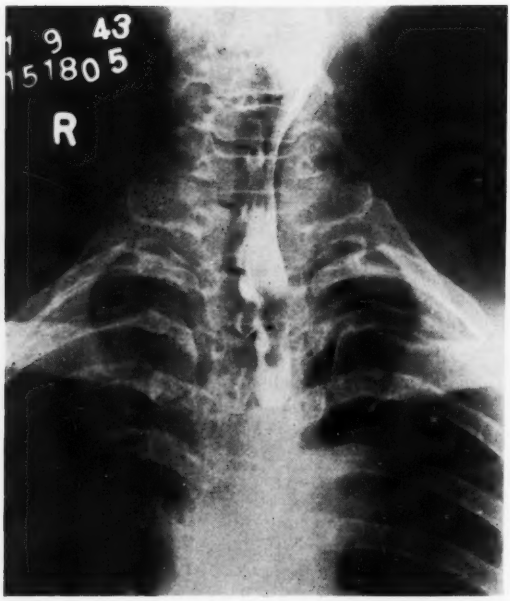

Fig. 2.—Roentgenogram, anterio-posterior view, showing the barium in the vallecula and the pyriform sinuses and the left side of the pharynx. The right side clears itself completely.

A neurological examination was requested and the report returned as follows: "the patient's gait is normal; her speech is hoarse; her general muscular strength is good. The examination showed no paresis or paralysis. She was attentive, cooperative and intelligent. The patient could report no change in mental state and said she had been working as usual. Examination of the cranial nerves was as follows: I, not tested; II, normal; fundi and veins normal; III, IV, V, VI, VII, normal; VIII shows diminished hearing on the left, the air conduction less than the bone conduction; IX and X, the palate deviates to the right with no gag reflex on the left. Aphonia is present. Abnormalities of the muscles of deglutition on the left were shown by x-ray films. XI and XII, normal. There is apparent involvement of the muscles of the neck. There is no vertigo or nystagmus. The reflexes are all bilaterally active and equal. Hoffman and Babin-

ski signs are negative. There is no ataxia, dysmetria, adiadokokinesis or rebound, nor any loss of vibrations or muscle sense.

"*Impression*: these findings fit into the so-called syndrome of Avellis; etiology probably vascular."

The prognosis is usually good in the syndrome of Avellis. In bulbar conditions the prognosis depends on the general prognosis; in myasthenia gravis it is unfavorable.

The internist and the neurologist should govern the treatment. Nutrition can be maintained by feeding with the stomach tube which meets with no resistance to its passage. If there is any contraindication such as ulceration of the esophagus, a gastrostomy should be done. Abundant supplies of fruit and vegetable juices, properly strained, are essential and it is the endoscopist's duty to see that they reach the stomach.

CONCLUSIONS

Given a patient with a high blood pressure, past middle life, in whom there is a sudden onset of symptoms such as has been described, that is, sudden difficulty in swallowing accompanied by a sudden change in voice, coupled with the physical signs noted, one should consider the possibility of the lesion being in the medulla.

However, there have been some cases of this condition reported in young children, so that age should not be one of the cardinal signs.

A competent roentgenologist as well as a competent internist will be of material assistance in diagnosing the condition.

Uusally these patients do not succumb to the primary lesion, but partially recover.

76 Bay State Road.

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XVIII

OSTEOMA OF THE MASTOID

J. W. Jervey, Jr., M.D.

Greenville, S. C.

The subject of osteoma of the mastoid has been mentioned recently by several authors. For the latest full account we are indebted to Simpson,[1] who in 1940 made a complete survey of the literature, found 30 cases and added two of his own. His article contains a complete bibliography. Since that time there has been one additional case reported by Cinelli[2] and another is presented here. There are now 34 cases on record. No doubt the actual number observed has been far greater, but the condition is relatively uncommon and worthy of mention. It is distinct, of course, from the exostoses of the bony canal which are of frequent occurrence. The following case is perhaps typical.

REPORT OF A CASE

W. S. was first seen in our office in October 1923, when he was about six years old. His tonsils were removed and he continued to have symptoms of sinus disease. After much treatment over a period of years and repeated advice to have a badly deflected septum corrected, he finally submitted in 1935 to a submucous resection. This gave complete relief of symptoms and he has had no upper respiratory difficulties since.

However, shortly after this he first noticed that a small pea-sized mass which had been observed for years over the right mastoid area was beginning to grow. About seven years after the submucous resection the growth appeared firm, rounded, and about the size of a five-cent piece. He was advised merely to have it kept under observation as there were no symptoms present. On November 4, 1943, he returned complaining of severe generalized headaches of several months' duration and the tumor appeared to be definitely growing. Hearing in the right ear was very poor, but our records and his own statement show that there had been no change in hearing for many years. Although our records go back to 1923 in this case there is no evidence that he ever had any middle ear disease. It is interesting to note that the right side of his face had always given a mild appearance

of paralysis but in actual fact no paralysis is or had been present. On November 4, 1943, the tumor appeared to have increased somewhat in size but no tenderness was present. X-ray films were taken and they showed that the growth involved only the outer plate. Removal was advised and the operation was carried out on November 17, 1943.

The skin and the periosteum were freely movable over the tumor which was densely adherent to the outer plate just above the knee of the sinus. Some of the mastoid cells were opened and were clean and healthy in every respect. It was found that the dense bone extended on down to the inner plate above, and in order to secure complete removal it was necessary to expose widely both the dura and the transverse portion of the sinus. The mastoid was not exenterated. The wound was closed after inserting a small drain and about three grams of sulfanilamide. The drain was removed in 48 hours and the wound healed by first intention.

So far he has had no recurrence of his symptoms. Although no careful check was made of his hearing before the operation, he states that the hearing since operation is exactly as it was before and as it has been for as long as he can remember. The pathological report was benign osteoma.

101 CHURCH STREET.

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XIX

CONGENITAL OCCLUSION OF BOTH ANTERIOR NARES

J. W. Jervey, Jr., M.D.

Greenville, S. C.

Congenital occlusion of the anterior nares, so far as I have been able to discover, is a rare condition, being much less common than occlusion of the choanae which in itself is so unusual that many men with long years of practice have never seen a case.

Discussions of etiology are rather unsatisfactory, it seems to me. Emphasis has been placed on the organization of secretions found within the nasal passages at and before delivery. However, I cannot conceive of organization of such material in the form of thin symmetrical membranes in the anterior nares occurring where epithelial surfaces are intact. My own thought, entirely without scientific investigation and proof, is that the condition must be the result of a faulty dissolution of tissue before birth, much the same as is found in the web so often met with in the lower end of the nasolacrimal duct in the newborn.

Reports on these cases seem to be somewhat vague and discussions of them are often found along with those of the posterior occlusions. The earliest case found was reported in 1908 by Craig[1] and was clear cut and to the point. It was handled much as was my own and with apparently good results. Other available accounts are rather unsatisfactory so that I do not know exactly how many cases are actually on record, certainly very few, as there are in existence only a handful of articles which even mention the subject. Most authors lead you to believe that complete surgical removal of the obstructive tissue and skin grafting must be employed. That may be necessary in some instances but was certainly not so in my case where methods were quite simple and entirely satisfactory.

REPORT OF A CASE

D. B. was first seen by me in January 1938 at the age of six months. She was brought in because the parents noticed that she never breathed through the nose. Contrary to the observations of most writers on the subject, she appears never to have had any great

difficulty with nursing and seemed to have automatically adjusted her behavior to the exigencies of her actual needs.

About one quarter inch in from each anterior naris there was a complete occlusion of the nasal passage. The nose otherwise appeared well formed and there was no other demonstrable abnormality. The choanae were not observed. The parents were requested to bring her back for further observation.

She was not seen again until August 1943, when she was five years old. At this time the occlusion in the right naris had apparently become absorbed so that a definite web structure was observed, in the middle of which was a tiny round opening about 2 mm. in diameter. The occlusion of the left side was still complete. The opening in the right was rapidly dilated to admit a No. 16 lacrimal probe, and a suitable piece of tight-fitting rubber tubing was pushed through and left in situ with a safety pin across the protruding end. This was taken out by the mother and cleaned daily and reinserted after mild anesthesia with pontocaine. The tubing was cut to about one and a half inches in length and the tip to be inserted was cut on a long level so that the piece could be inserted easily and with increasing pressure as it was pushed in to the limit. As the opening in the web became larger, a tube of larger size was substituted so that in a month's time the vestibule opening was approaching normal size.

At this period, under light general anesthesia the web in the left naris was split vertically with a submucous knife which went readily through into the nasal passage. There was very little bleeding. The web was about one-eighth of an inch thick or less, and the interior of the nose appeared normal in every way. The remaining web on the right was then cut through vertically above and below. Each naris was packed firmly with a finger cot filled with vaseline gauze packing. This was left in situ for six days as I wished granulations to form so that there would be no pain on removal. There was no trouble at removal, nor has there been at any other time. The packs were replaced by a rubber tube in each nostril with one safety pin through both protruding ends. This device through which she breathes easily is worn at night and dispensed with during the day. She was seen last on December 26, 1943, five weeks after operation, at which time both passages were virtually normal in size and appearance and there was no granulation tissue present.

101 CHURCH STREET.

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Society Proceedings

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

Meeting of Monday, November 1, 1943

THE PRESIDENT, DR. SAMUEL J. PEARLMAN, IN THE CHAIR

Vitamins in Otolaryngology

H. B. PERLMAN, M.D.

(Abstract)

In looking for a relationship between clinical otolaryngology and vitamin physiology the soundest approach is to study the clinical and pathologic picture of known deficiency states in man. If there is no evidence of ear, nose and throat disease in a known deficiency state, it does not seem reasonable to expect benefit from the treatment of ear, nose and throat disease with the particular vitamin.

While vitamin A deficiency in man has been widely studied, no characteristic otolaryngological signs have been reported except an occasional case of metaplasia of the respiratory mucous membrane in children dying with this disease.

Thiamin deficiency in man has been carefully studied. No otolaryngological signs are reported.

Nicotinic acid deficiency does produce characteristic lesions of the mouth, tongue and esophagus along with the other classical findings. Redness, dryness, atrophy and small ulcers characterize the mucous membrane changes of interest to the otolaryngologist. These lesions respond to nicotinic acid therapy.

Riboflavin deficiency has also been carefully studied. Cheilitis and ragades at the corner of the mouth are characteristic findings. Before these lesions develop, burning of the mouth, tongue and eyes is noted and these respond to riboflavin therapy.

Ascorbic acid deficiency produces perifollicular hemorrhage and delayed wound healing only after prolonged, complete depletion.

Vitamin D deficiency is not associated with any characteristic lesion of the ear, nose or throat.

Vitamin K deficiency causes a low prothrombin level in the blood and is important in patients with obstruction of the bile ducts and in the newborn.

In contrast to this limited relationship between known vitamin deficiency states in man and clinical otolaryngology, many laboratory experiments on animals show suggestive relationships. An important example is the effect of vitamin A depletion in young animals on the length and size of the internal auditory meati. Marked lengthening and narrowing by new bone take place. This may lead to impaired auditory nerve functioning and to changes in the end organs. No direct clinical importance can be attached to this experiment. Classical signs of vitamin A deficiency are practically unknown in this country and in those countries where the disease is commonly reported this bony hyperplasia or functional impairment is not seen. Until more is known about the physiology of vitamins and more widely accepted criteria are developed for recognizing a deficiency state, further co-relations with otolaryngology will be difficult.

DISCUSSION

DR. HOWARD C. BALLENGER: I had hoped Dr. Perlman would talk a little more about a possible vitamin A deficiency in chronic sinusitis. Perhaps he can elaborate somewhat on that subject.

DR. THOMAS C. GALLOWAY: Dr. Perlman's careful study merits close attention but I think he is a little more discouraging than he might well be. It may not be quite valid to say that if changes demonstrable in the laboratory are not established with extreme depletion of the vitamins, we can assume that there are no subclinical changes with lesser deprivation. That may partly depend upon doubtful criteria. For instance, lesions about the mouth are taken as an index for riboflavin deficiency. However, there are other less obvious findings in riboflavin deficiency, such as vascularization of the cornea which can be picked up by the slitlamp. This finding, associated with definite ocular discomfort, may be reversed by administering the vitamin and relief of the patient's symptoms will result.

Similarly with vitamin A deficiency, certain clinical states are suspect. A condition that is rather prevalent is itching eyes, especially in dieting women or in patients with gallbladder disease whose

food contains few sources of vitamin A. With the slitlamp there may be seen edema and changes in the corneal epithelium and, in extreme cases, keratomalacia. There may be associated skin changes including keratoses and even a tendency to formation of small basal cell carcinomata. This condition in some cases seems to respond to vitamin A administration.

I think it is important in the laboratory to expect definite findings if we are going to have positive pronouncements. On the other hand, we cannot ignore the factor of subclinical disturbances which may respond to therapy even though this may lead to some exploitation of the use of vitamins.

DR. A. R. HOLLENDER: The general concensus seems to be that if an individual is deficient in one vitamin he is probably deficient in more than one. I happen to be familiar with a certain study now being conducted in the East. Large doses of vitamin A are being administered for certain types of deafness, particularly those which have an associated tinnitus. As much as 100,000 units are given daily with results that seem highly encouraging, especially in controlling the tinnitus. No report has been made as yet, nor are any claims made concerning this therapy. While vitamin A has been employed in cases of hearing defects, the large dosage now advocated has not heretofore been suggested. There remains to be established a rationale for this treatment. Another problem is the perfection of a product with which the dosage can be properly controlled.

While the essayist's theme is primarily vitamin A, I should like to bring up a point in connection with nicotinic acid. Most of us are familiar with Atkinson's work in Ménière's disease. If Dr. Perlman's theory is correct, that the results from nicotinic acid are purely transitory, Atkinson's suggestions concerning nicotinic acid therapy should be discredited. Whether results with nicotinic acid are attributable to regulation of other existing vitamin deficiencies I do not know. We do know that administration of thiamin hydrochloride, for instance, influences other vitamin factors. Not many years ago the Council on Pharmacy spoke unfavorably of vitamin therapy. The situation has changed. In a recent report the administration of multiple vitamins is no longer regarded as altogether irrational, if it appears that a deficiency of more than one vitamin exists and it is impossible to determine which are deficient.

DR. IRVING S. CUTTER: I was tremendously impressed with this paper. I am sure Dr. Perlman has followed the work of certain men who are observing the catalytic action of certain vitamins,

wherein they appear to exert synergistic effects. Particular fractions appear to possess the power to speed up reactions. This may explain in part clinical effects which may not coincide exactly with laboratory data.

DR. WALTER THEOBALD: Dr. Perlman is to be commended for having taken a preparation which is handed out at every drug store, given away in samples, and boosted over the radio and, with this, developed this excellent presentation. Parts of his paper are rather discouraging. Empirically, we are all giving vitamins and I had hoped to learn whether I should give vitamins for degenerative nerve conditions of the ear. I have tried it in large doses and have obtained what I thought were favorable results in some few cases. Whether the results are due to the vitamin, to psychology or to God, I cannot say, but now and then good results are obtained. I wish he could tell us the effect of vitamins upon the bone of the cochlea in the human, if any.

DR. H. B. PERLMAN (closing): Dr. Ballenger asked about the relation of vitamin A deficiency to sinusitis. There are reports of autopsies on children dying with xerophthalmia. There is an occasional instance of metaplasia of the respiratory mucous membrane from columnar ciliated to pseudostratified. I had occasion to talk with some of the men from South America who attended the last Academy meeting about this problem. They see many classical deficiency states and associated with the classical findings there may often be sinusitis or otitis media. I asked if they could recognize the deficiency state by these findings alone and they said they could not. Remember further that vitamin A deficiency as shown by xerophthalmia is practically unknown in this country.

Dr. Galloway spoke of the vascularization of the cornea as a sign of riboflavin deficiency. That is one of the more recent attempts at early recognition of a deficiency state. Another method used is to determine the amount of a given vitamin that is kept in the body after feeding a known amount, and how much is excreted. Criteria for recognizing a deficiency state are still in the process of formulation. Some are widely accepted, others are open to criticism. Slit-lamp findings in the eye might be checked against some more quantitative method. The qualitative determination of vitamin A in the plasma of a depleted animal shows clear-cut marked reduction over the controls. Such chemical methods are probably least open to criticism. Correlation studies between biophotometer tests and the level of vitamin A in the plasma have as yet given no conclusive results.

As to the relation between the hormones and the vitamins little is known. Some of the studies in our field that have been published entailed the use of both vitamins and hormones in a single patient. It was practically impossible to arrive at any conclusion as to what, if anything, was having an effect. Hormone deficiency states are also often hard to define.

Dr. Hollender mentioned the use of very large amounts of vitamin A in deafness. Little is known about the physiology or pharmacology of massive doses of vitamins. Some vitamins are harmless in overdoses, usually being promptly excreted. Others may be harmful in massive doses. I am not sure what place nicotinic acid has in the treatment of Ménière's disease. Perhaps some acute episodes are being reversed by its use as a vasodilator.

Dr. Cutter mentioned the catalytic action of the vitamins. Information about the biochemical properties of vitamins has grown rapidly. The experimental literature is filled with such reports. Riboflavin is a widespread catalyst for cell oxidation. Synthesis of vitamins in the body and interaction of one vitamin with another and many other basic observations are being made. However, as clinicians, we must keep a proper perspective when attempting to find a new therapeutic approach.

Dr. Theobald asked about large doses of vitamins in nerve deafness. I do not know the rationale or the results of such treatment. As to the effect of vitamins on the cochlear capsule, there is no doubt that experimentally one can produce this large amount of new bone in the internal meatus by vitamin A depletion as pointed out in the paper. However, there is little clinical significance in such an observation.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

Meeting of Monday, December 6, 1943

THE PRESIDENT, DR. SAMUEL J. PEARLMAN, IN THE CHAIR

Terminal Stages in the Development of the Human Stapes

BARRY J. ANSON, PH.D.

EARL W. CAULDWELL, M.S.

ARTHUR F. REIMAN

(This paper appears on page 42 of this issue)

Muscular Variations of the Pharyngeal-Esophageal Segment

NORA B. BRANDENBURG

(Abstract)

The pharyngo-esophageal segments in 41 bodies were studied. Attention was directed to the muscular coat surrounding the esophagus, especially to the so-called esophageal triangle located below the cricopharyngeus. Separation between the pharynx and the prevertebral muscles was easily accomplished in all cases.

The majority of the inferior constrictor fibers were seen to ascend obliquely and insert into the median raphe. The most inferior fibers coursed horizontally and overlapped the beginning of the esophagus. The direction of their fiber arrangement was directly opposite to that of the pharyngeal muscles. Variations occurring in these structures result in marked variation in the muscular protection of the pharyngo-esophageal segment.

Hernias in general bear a direct relationship to tissue integrity and muscular protection. With muscular variations there is an additional contributing factor to hernial formation. Correlation of the muscular variations and deficiencies to different types of hernias in other regions has been established. It is believed that a pulsion divert-

iculum of the esophagus bears the same direct relationship to congenital muscular variations of the pharyngo-esophageal segment and variations in Laimer's triangle as does inguinal hernia to the triangle of Hesselbach. There may be not only a variation in the number of muscle fibers attaching into the tendon of Gillette, but a variation in the distribution, quality and number of muscle fibers diverging over the posterior surface of the esophagus. This muscle fiber diversity results in alterations of size, shape and configuration of Laimer's triangle, thus making it a vulnerable point for herniation.

DISCUSSION

Dr. Glenn J. Greenwood: Dr. Brandenburg is to be congratulated upon this interesting work. It was my privilege to read the manuscript. I wonder what the relative percentage of variation was found to be.

In 1939 I saw a case of acute diffuse suppurative mediastinitis secondary to traumatic rupture of a diverticulum of the cervical esophagus. The case went on to fatal termination in 13 days. Of interest is the pathologist's report on the esophagus, to the effect that, "On the right lateral wall just a little below the level of the cricoid cartilage there is a diverticulum 1 cm. in diameter and 1 cm. long. Its thin, white floor contains a small perforation." Dr. Brandenburg's described muscular variations of the pharyngo-esophageal segment present an hypothesis which might help to explain the position of this pouch.

Dr. Paul Holinger: The problem presented in Dr. Brandenburg's thesis is not only of academic importance but of practical importance as well. We all know that clinically most diverticula occur to the left. I would like to ask her the percentage anatomically in which the muscular dehiscence was found on the left. Practically, of course, the findings reported explain the lodgment of foreign bodies in this area, the dehiscence accounting for the ease of perforation of the upper esophagus. One acquires a great deal of respect for this area in viewing the complete absence of musculature in the upper portion of the esophagus as shown in Dr. Brandenburg's illustrations. One wonders why perforations do not occur more frequently in this locality when one sees the large portion of the esophageal ring covered only with mucosa.

Dr. Alfred Lewy: I would like to ask if Dr. Brandenburg has any information as to the possibility of local factors causing

spasm of the cricopharyngeal muscle in addition to the ones she described.

DR. THOMAS C. GALLOWAY: This is a fine piece of work; it has been very carefully performed and should be valuable.

In surgery of diverticula of the esophagus the important thing is straightening the angle where the diverticulum meets the main esophagus, so one can really lift the esophageal pouch upward and straighten it out to form a nearly straight line with the descending esophageal wall. It has been generally assumed that these diverticula begin above the cricopharyngeal muscle and hang over that as a shelf; and if there has been inflammation in the diverticular sac there are adhesions of that angle. The difficult part of surgical removal of a diverticulum, whether in one or two stages, is freeing of the angle. I think it very important to have established just where the pathologic process starts, whether above or below the cricopharyngeal junction. If a diverticulum comes from any defective part of the upper esophagus, with herniation involving the mucosa and submucosa, the dissection is probably much less safe than we have assumed. I shall approach that angle dissection with considerably more trepidation than heretofore.

DR. NORA B. BRANDENBURG (closing): Dr. Greenwood asked the percentage of triangular variations as seen in my specimens. The specimens varied as much as did the noses of the 41 bodies examined. The triangles were of different measurements in most of the specimens. Only the gross anomalies were shown in my paper. In order to make a definite percentage of those to the right or to the left, I would have to establish a basic triangle, which I was unable to do. The largest triangle was 1.5 cm. across the base and 6 cm. on each side.

Dr. Holinger asked about the variations of the triangles to the left. I have no data on that, but I do not think it bears much relationship to the fact that diverticulum sacs are found most often on the left.

In answer to Dr. Lewy's question whether this approach would explain the Vinson-Plummer syndrome, there are no factors in my research problem that would explain this syndrome.

Books Received

The Head and Neck in Roentgen Diagnosis.

By Henry K. Pancoast, M.D., Late Professor of Radiology, University of Pennsylvania; Eugene P. Pendergrass, M.D., Professor of Radiology, University of Pennsylvania; Director, Department of Radiology, Hospital of the University of Pennsylvania; and J. Parsons Schaeffer, M.D., Ph.D., Professor of Anatomy, Jefferson Medical College; Director, Daniel Baugh Institute of Anatomy, Jefferson Medical College. Pages xxxii + 944, with 1251 illustrations. Springfield, Illinois, Charles C. Thomas, 1943. ($6.50).

This monumental volume which first made its appearance in 1940 is now in its second printing. The text is all that one could expect even from its three distinguished authors.

Besides a profusion of roentgenograms and illustrations showing in detail the various positions and angulations required to demonstrate lesions of the head and neck, there are accompanying anatomical descriptions, diagrams, and drawings.

All illustrative material is reproduced with great fidelity, which in the case of roentgenograms requires more than the usual gradation of light and shade.

The subject matter is too detailed, varied and extensive to permit of discussion here. The most remote conditions are adequately handled.

The book should be on the shelf—better the desk—of every otolaryngologist.

Clinical Audiometry.

By C. C. Bunch, M.A., Ph.D., Formerly Associate Professor of Otology, Medical School, University of Iowa; Associate in Research Otology, Johns Hopkins University; Professor of Applied Physics of Otology, School of Medicine, Washington University; Associate Director of Central Institute for the Deaf, St. Louis; Research Professor in Education of the Deaf, School of Speech, Northwestern University. Pp. 186, with 74 illustrations. St. Louis, The C. V. Mosby Company, 1943. (Price $4.00).

To anyone who knew the author of this monograph and had the privilege of watching him at work it will be axiomatic that a book from his pen is characterized by thoroughness, completeness and a punctilious regard for detail.

From 1917, when he received the degree of Master of Arts from the University of Iowa, until the time of his death which occurred

only a day or two after this manuscript was completed, Bunch devoted his life to the study of audiometry and its application to clinical practice. In 1919 in collaboration with L. W. Dean, he presented the first continuous pitch-range audiometer before the American Otological Society. This was a generator consisting of a disc with teeth which rotated in a magnetic field at controlled speeds to produce sounds of the required pitch in a telephone receiver.

With the advent of vacuum-tube oscillators his interest turned to the compilation and interpretation of audiograms. Probably his most important contribution was the study and description of curves in traumatic deafness resulting from occupational injury.

This monograph constitutes a manual of the use of the audiometer, including technical phases, the building of a soundproof room and the testing of school children. The section on interpretation of curves is complete and informative and contains many examples from Bunch's extensive collection.

There are sections on the use of residual hearing, the function of the audiometer in selecting a hearing aid, and finally, a historical review of instruments in use from 1882 to the present time.

Transactions of the American Laryngological Association.

Volume 65, 1943, octavo, 193 pages.

This current volume of the Transactions is of interest in that it includes an index of subjects and authors of all papers presented before the American Laryngological Association from its inception in 1879 to the present time.

Office Treatment of the Nose, Throat and Ear.

By Abraham R. Hollender, M.C., M.D., F.A.C.S., Associate Professor of Laryngology, Rhinology and Otology, University of Illinois, College of Medicine; Otolaryngologist, Research and Educational Hospitals, Chicago, Illinois. Octavo, pp. 430, illustrated. Chicago, The Yearbook Publishers, 1943. (Price $5.00).

This excellent manual, as its title indicates, deals exclusively with treatment. By avoiding entirely the mention of symptomatology and pathological considerations the author very properly implies that the reader is familiar with these subjects before beginning treatment; he does not fall into the error of dealing with them superficially as he might well have done in a book on treatment, thus tempting the immature reader to be satisfied with insufficient knowledge of them.

The field is well covered, the material is judiciously handled and the illustrations are excellent. Complete references accompany each chapter.

This book should be extremely useful to house officers and others beginning the practice of otolaryngology. Its completeness and accuracy in the description of methods should recommend it also to the more experienced.

Oral Pathology. A Histological, Roentgenological, and Clinical Study of the Diseases of the Teeth, Jaws, and Mouth.

By Kurt H. Thoma, D.M.D., Professor of Oral Surgery and Brackett Professor of Oral Pathology, Harvard University; Oral Surgeon and Chief of Dental Service, Massachusetts General Hospital; Oral Surgeon to Brooks Hospital; Dental Surgeon to Dental Department and Consultant in Oral Surgery to Tumor Department, Boston Dispensary and Joseph H. Pratt Diagnostic Clinic; Consulting Oral Surgeon, New England Baptist Hospital; Consulting Oral Surgeon, Beth Israel Hospital. Second Edition. Pp. xxi + 1328, with 1388 illustrations including 128 in color. St. Louis, The C. V. Mosby Company, 1944. (Price $15.00).

This impressive work, now in its second edition, is devoted entirely to the anomalies, maldevelopments, and diseases of the teeth, jaws and mouth. The presentation is masterful and the illustrations are suberb. It is so well done that it will render any other work on the subject superfluous for some time to come.

Approximately one-third of the book is devoted to purely dental matters which may not directly concern the otolaryngologist. However, the remaining two-thirds contains much that is of practical interest to any medical man, material that will not readily be found in any other type of text. Author and publisher are to be congratulated upon this fine volume.

Diseases of the Ear, Nose and Throat.

By Douglas G. Carruthers, M.B., Ch.M. (Sidney), F.R.A.S.C., Honorary Ear, Nose and Throat Surgeon, Sidney Hospital and Eastern Suburbs Hospital, Sidney; Consulting Ear, Nose and Throat Surgeon, Canterbury District Memorial Hospital, Sidney. Pp. 397, with 133 illustrations. Sidney-London, Angus and Robertson, Ltd., 1943.

This is an attractive little book, well written and well illustrated.

Beyond this, however, there is little to distinguish it from a long line of similar manuals. In the opinion of this reviewer too much stress is laid upon surgery and not enough upon the indications for it. A good list of competent references would greatly enhance the value of this volume.

Rehabilitation of the War Injured.

A Symposium. Edited by William Brown Doherty, M.D., and Dagobert D. Runes, Ph.D. Pp. 684, illustrated. New York, Philosophical Library, 1943.

Similar in format and treatment to a previous volume on *War Medicine*, this collection of papers deals with the rehabilitation of the war injured in all its phases. The list of authors contains many distinguished names and the approach is extremely practical.

The first section, headed "Neurology and Psychiatry" deals with the psychological reactions to injury, resocialization and the rehabilitation of the individual suffering from head injuries and other major war wounds. There follows a section on "Plastic Surgery and Its Fundamentals." It deals with flaps, scars, grafts and implants and the relation of the early care to the final outcome of major face wounds in war surgery.

The third section deals with "Orthopedics:" amputations, stumps, and transplantations. The subsequent sections are devoted to "Physiotherapy," "Occupational Therapy," "Vocational Guidance," and "The Legal Aspects of Rehabilitation."

The illustrative material is excellent but the reproductions are, on the whole, poor.

Abstracts of Current Articles

NOSE

Patulin and the Common Cold.

Brit. M. J., Nov. 27, 1943, p. 683.

Patulin, the antibacterial derivative of the mould *Pencillin patulum* Bairier, was studied by Raistrick and his colleagues (*Lancet* Nov. 20, 1943, p. 625). It was found to be toxic: 0.5 mg. 20 g. intravenously kills 65% of mice, and a concentration of 1 in 2000 inhibits phagocytosis. In concentrations between 1 in 33,000 and 1 in 100,000 the growth of various bacteria, gram-negative as well as gram-positive, was inhibited. Strengths of 1 in 5,000 or 1 in 10,000 are recommended for therapeutic use.

Preliminary observations of its therapeutic value in the intranasal treatment of common colds by W. E. Gye are confirmed by W. A. Hopkins. Cold sufferers were impartially treated in two groups: one with a solution of patulin in phosphate buffer, and a control group with phosphate buffer alone. Classification as "cured within 48 hours" or "not cured" gave the mean figures of 58% cured in the treated group and 9.4% in the control group. The common experience was six hours complete relief after one application, and four-hourly application was all that was found necessary.

GODWIN.

PHARYNX

Recognition and Radium Treatment of Nasopharyngeal Lymphoid Tissue.

Fisher, Gilbert E.: South. M. J. 36:702 (Oct.) 1943.

Proliferation of the nasopharyngeal lymphoid tissue following adenoidectomy and tonsillectomy is discussed and a method of applying radium to the nasopharynx described.

Two hundred fifty cases of postnasal discharge so treated were examined six and twelve weeks after treatment with the following results:

(1) Two hundred thirty-four patients reported their postnasal discharge had ceased.

(2) Direct nasopharyngoscopy revealed diminution in the size of the lymphoid tissue in all these cases.

(3) Excessive dryness of the nasopharynx was not noticed in a single case.

(4) Improvement of hearing, as measured by audiometric studies before and after treatment, was found in 37 cases of conductive deafness caused by long standing obstruction of the eustachean tubes.

(5) Sixteen cases were unimproved by treatment. Eleven of these suffered from seasonal allergic rhinitis. Six were definitely improved following a second radium treatment, while the other five were unimproved.

(6) Three of four patients with perforated tympanic membranes and mucoid discharge had dry ears after five weeks; one required nine weeks.

(7) Eleven patients complaining of tinnitis were unimproved except that their postnasal discharge ceased.

SOOY.

TRACHEA

Carcinoma of the Trachea; Report of Two Cases Diagnosed and Treated by Roentgen Rays.

Brown, Samuel, Weiss, H. B., Iglauer, Samuel, and Fine, Archie: Radiology 41: 394-397 (Oct.) 1943.

Two cases are reported in which radiographs of the chest in the oblique and lateral positions with a barium filled esophagus revealed a mass between the trachea and the esophagus. The mass caused a filling defect of the esophagus in one case and a displacement of the esophagus in the other case. Endoscopy with biopsy revealed squamous-cell carcinoma. One case is alive three years later and is in good health without symptoms (cough, expectoration of blood or bloody mucus, manubrial pain). The second case died of heart failure within seven months. Intensive x-ray therapy was the only method of treatment used. Exact localization of the growth is imperative in administering the therapy.

JORSTAD.

Sealing Intratracheal Catheters, and a Listening-tube.

Pinson, K. B.: Brit. M. J. Dec. 11, 1943, p. 747.

Sealing of an ordinary Magill catheter for intratracheal anesthesia to prevent leakage of cyclopropane and allow control of intrathoracic pressure is accomplished by the writer by tying the chin back with two turns of bandage. With the phenominal relaxation of the jaw under cyclopropane the soft parts about the glottis and the base of the tongue are approximated to the posterior pharyngeal wall, providing sealing without the use of packs or an inflatable cuff. This method is not recommended in operations about the mouth. In some patients the face becomes congested.

The use of a small T-connected listening-tube from the Magill connection to the anesthetist's ear is suggested to facilitate hearing the patient's breathing.

GODWIN.

ESOPHAGUS

Roentgenologic Aspects of Acute and Chronic Esophagitis.

Paul, Lester W.: Radiology 41:421-430 (Nov.) 1943.

Clinical and radiographic study of five cases of acute ulcerative and chronic esophagitis are presented. Acute ulcerative esophagitis is rather uncommon as a clinical disease, is most often associated with peptic ulcer or develops during the immediate period following upper abdominal operations. Anything which permits gastric juice to come in contact with the esophageal mucosa may predispose to its development. Roentgen changes consist of severe spasm of the distal esophagus, loss of mucosal folds, and a fine roughening of the surfaces. The lesion tends to progress to fibrous stricture. Chronic esophagitis severe enough to cause symptoms is uncommon. A diffuse fibrous stricture results. In other instances roentgen manifestation is intermittent; there is diffuse spasm of the lower half or third, with thickening of the mucosal folds.

JORSTAD.

BRONCHI

Bronchoscopy and Asthmatoid Respiration.

Friedberg, Stanton A., Capt., M.C., A.U.S.: J. A. M. A. 123:85-87 (Sept.) 1943.

The importance of bronchoscopic investigation in all cases of a typical bronchial asthma is emphasized.

"Asthmatoid wheeze" is described as an expiratory wheeze similar to, but drier than, an asthmatic wheeze, and is postulated to arise

from contraction and narrowing of the bronchial lumen about some abnormality during expiration.

Illustrative cases are presented of asthmatoid respiration due to: Popped corn hull in the right main bronchus, pistachio nut shell in the trachea, sizable toy dog in the upper esophagus, degenerated iron tack in the left main bronchus, anaerobic abscess in the left upper lobe, stenosis of the left lower lobe bronchus, and tuberculosis of the left main bronchus.

SOOY.

EAR

Moniliasis of the External Ear Canal.

Dobes, William L.: South. M. J. 36:614 (Sept.) 1935.

From a review of the literature the author presents evidence that Monilia albicans is not found on normal skin but has been recovered from the feces of normal persons. It has also been found pathologically in dermatoses, in bronchopulmonary infections, and in some cases of osteomyelitis, glossitis, stomatitis, vaginitis, paronychia, and onychia.

Only two references to involvement of the external ear canal by Monilia albicans were found. These were successfully treated by mercuric iodide in alcohol, and mercuric chloride in alcohol respectively.

A case of bilateral external otitis is presented from which Monilia albicans and Aspergillis fumigatus were isolated.

Treatment consisted of filling the external ear canal once daily with a two per cent solution of gentian violet for five to ten minutes. Healing was complete within ten days.

The gross and microscopic appearance of the fungus and methods of culture are described.

SOOY.

MISCELLANEOUS

Diagnosis of Lipoid Pneumonia by Aspiration Biopsy.

Nathanson, L., Frenkel, D., and Jacobi, M.: Arch. Int. Med. 72:627, 1943.

Lipoid pneumonia is a chronic inflammatory condition of the lungs that is produced by the inhalation of oils. These are usually given as medication by mouth or as drops into the nose and in very

young or old patients the oil may go down the trachea rather than the esophagus. The authors of this paper point out that this condition has been reported repeatedly but only rarely has the diagnosis been made antemortem.

The clinical diagnosis rests on the history of administration of oils, infiltrations in the lower fields of the lung as shown by x-ray examination, and improvement after discontinuance of the oils. The work reported here consists of the recognition of this condition by aspiration biopsy of the lung. A long needle is introduced directly into the lung through the chest wall and the material withdrawn with suction. Smears are made from the small amount of material in the needle and macrophages containing phagocytized lipoid recognized. Although the technique is described well, there is only little discussion of its possible dangers.

HARFORD.

Actinomycosis of the Tongue Successfully Treated by Sulphonamides.

McCloy, A.: Brit. M. J. July 24, 1943, p. 106.

A case of actinomycosis of the tongue in a 59-year-old farmer is presented.

Potassium iodide was tried but stopped after ten days due to iodism. Three successive four-day courses of sulphapyridine were then given, following which the patient has remained clinically well for two years.

SOOY.

Faucial and Labial Diphtheria.

Anderson, Manual: Brit. M. J. July 24, 1943, p. 104.

The rarity of diphtheritic lesions on the lips is shown by the fact that there were only 49 instances reported in 7314 fatal cases of diphtheria in Hamburg.

A case of a ten-year-old girl is presented and illustrated. This patient accidentally bit her lower lip during the first few hours of an attack of faucial diphtheria, and in 24 hours developed a local membrane from which positive cultures were obtained.

All lesions cleared six days after the administration of antitoxin.

SOOY.

Transfusion Reactions and Fatalities Consequent on Circulatory Overloading.

Drummond, R.: *Brit. M. J.*, Sept. 11, 1943, p. 319.

Normal subjects according to experimental work quoted can tolerate up to 2000 cc. of saline, serum, or blood intravenously in as little as 15 minutes without symptoms. Compensation is accomplished by dilation of the heart and pulmonary vessels.

Caution is urged in transfusing patients with chronic anemia or myocardial damage.

Two fatal cases are reported from too rapid transfusion in which the initial hemoglobins were 25 per cent and 22 per cent, respectively. In one instance 120 cc. was given in 10 minutes and in another, 540 cc. in 30 minutes. Necropsy showed pulmonary edema and congestion.

Similar caution is urged in regard to large transfusions and two illustrative cases and a fatality are recorded.

A transfusion rate not in excess of 1 cc. per lb. of body weight per hour is advised, and in chronically anemic patients with a hemoglobin percentage of 25 or less the rate should be halved.

Transfusion with universal donor or type 0 blood is discouraged particularly in anemic patients since the ratio of donor's serum to recipient's cells is abnormally high.

Frequent observation of the recipient and prompt cessation of the transfusion with the onset of symptoms of pulmonary congestion are advised. Oxygen, morphine and atropine, and venesection may also be used but adrenaline is condemned.

A discussion by Stallworthy (Brit. M. J., Oct. 2, 1943) urges similar caution in giving transfusions to patients who have had a hemorrhage and who have an associated toxemia.

Sooy.

Ear, Nose and Throat Casualties in a General Hospital in the Middle East.

Collins, E. G., Major, R.A.M.C.: *Brit. M. J.*, Sept. 25, 1943, p. 386.

A review and statistical analysis of 1054 patients with ear, nose or throat complaints is presented.

War injuries were exceedingly few in number.

The commonest diseases were acute tonsillitis, otitis media, otitis externa and sinusitis.

A plan for prophylaxis and treatment of each of these catagories is outlined. Conservatism is favored over surgery in most instances.

SOOY.

The Treatment of War Wounds with Penicillin.

Garrod, L. P.: Brit. M. J. Dec. 11, 1943, p. 755.

Prof. L. P. Garrod gives an account of the report published by the War Office on the use of penicillin in war wounds in the North African and Sicilian campaigns under the direction of Prof. H. W. Florey and Brigadier Hugh Cairns.

Penicillin was applied locally as a solution in distilled water containing 250 units of penicillin per c. cm., as a powder in which calcium penicillin was diluted with sulfanilamide, and as a cream in a lanette base. Systemic treatment was given by intramuscular or continuous intravenous injections of sodium penicillin in daily doses of about 120,000 units.

In the local treatment of chronic wounds of three weeks' to four months' duration by solution, powder, and cream, it was found that accessible surfaces could be sterilized but that deep-seated infection could not be controlled.

Eight chronic cases of septic compound fracture were treated by intensive systemic administration of penicillin with dosage over 1,000,000 units in four of the cases. The infection was cleared in six of the eight cases.

One hundred seventy-one recent soft-tissue wounds mostly of three to twelve days' duration were treated by penicillin applied locally and by immediate closure. Three to 10 c. cm. of penicillin (250 units per c. cm.) were injected twice daily for four days into the wound through 1/8-inch rubber tubes introduced through stab holes or through the wound itself. In a few cases local application of powder with immediate closure was used without irrigation. Complete union was secured in 104 cases, sub-total union (i. e. healing by granulation in some part of the wound) in 60, and failure of healing in 7. The wounds showed little reaction, remaining dry or discharging a thin "gram-negative pus" containing Ps. pyocyanea which did not prevent rapid healing.

Recent compound fractures (5-14 days old) were converted to simple fractures by closure and treated systemically with 100,000 units of penicillin daily for five days. Of 31 such cases complete union was achieved in 16, subtotal in 10, and 5 were failures. The worst results in fractures of the femur were attributed to inadequate dosage and too short a course of treatment. In two cases penicillin-resistant cocci were encountered.

Infected brain wounds 3-12 days old were excised, cleaned, and closed, leaving a small tube through a stab wound into the brain cavity. Pus was aspirated and penicillin solution injected through this tube twice daily for three to six days. Of 23 cases so treated only three died.

GODWIN.

Facial Fractures as Seen in the Naval Service.

Lipscomb, Thomas H.: South. M. J. 36:665 (Oct.) 1943.

The method of production of facial fractures in airplane and automobile accidents is discussed.

Hemorrhage, tissue separation, and shock are treated promptly. Following this a more thorough examination is carried out including x-ray films of the skull. Bucky-Water's, occlusal or zygomatic views are taken if necessary.

The importance of teamwork between the orthopedist, oto-laryngologist, dentist, and anesthetist in arriving at a decision as to treatment is stressed.

An effort is made to obtain: satisfactory occlusion, satisfactory vision, drainage of hemorrhage or pus, and a good cosmetic result.

Excellent illustrations of an airplane instrument panel before and after a crash, and of a patient with multiple facial fractures treated by pin fixation accompany the article.

SOOY.

Notices

GRADUATE COURSES IN OTOLARYNGOLOGY

The Indiana University School of Medicine offers its annual Course in Anatomical and Clinical Otorhinolaryngology, April 17-29 inclusive. The lectures will be given by the staff of the department. For further information write Dr. C. H. McCaskey, Department Chairman, 608 Guaranty Building, Indianapolis 4, Indiana.

The University of Cincinnati College of Medicine offers its Eighth Annual Graduate Course in Otolaryngological Surgery on the Cadaver, May 15-20, inclusive, 1944.. Kindly address all communications to The Dean, College of Medicine, Eden and Bethesda Aves., Cincinnati 19, Ohio.